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MYALGÍA OF THE PHARYNX

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As an addition to a study of myalgia of the head which we have published elsewhere,[1] we wish to present this report of a syndrome of myalgia of which a fairly extensive search of the literature has failed to furnish a description. This syndrome we wish to designate "myalgia of the pharynx." While this syndrome often is associated with myalgia elsewhere, it may occur independently, and when it does, it may present a puzzling diagnostic problem. Patients who have myalgia of the pharynx present themselves with the complaint of a severe sore throat, marked pain on swallowing and occasional attacks of hoarseness. This complaint has been present for several months to several years. On inspection none of the usual signs of inflammation of the upper part of the respiratory tract are present, especially inflammation of the mucous membrane. The nasopharynx does not show any evidence of change, the laryngeal mucosa is of normal color and the vocal cords move normally. Unless palpation of the pharyngeal and laryngeal structures is resorted to, the diagnosis may be overlooked easily. Indeed, the chief complaint of these patients is likely to be that they have visited many physicians who refused to credit their story of sore throat and strongly intimated that the complaint was psychogenic. This diagnosis is the more readily arrived at because these patients present the background of most forms of myalgia, that is, so-called sympathetic imbalance, with coldness and mottling of the hands and coldness of the feet, usually associated with chronic nervous exhaustion and an anxiety tension state.

The diagnosis becomes clear to the clinician who will take the trouble to palpate the pharyngeal and laryngeal structures, as patient after patient

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From the Section on Otolaryngology and Rhinology (Dr. Williams) and the Section on Physical Therapy (Dr. Elkins), Mayo Clinic.

1. Williams, H. L., and Elkins, E. C.: Myalgia of the Head, Arch. Phys. Therapy **23**:14-22 (Jan.) 1942.

presents the identical points of tenderness, sharply localized to certain muscle groups and attachments:

1. Unusual and exquisite tenderness is present over the styloid process and over the tendinous loop of the insertion of the digastric muscle to the body of the hyoid and the insertion of the stylohyoid muscle at the junction of the body and greater cornu of the hyoid bone. Often the stylohyoid and the posterior belly of the digastric muscle are tender and tense and can be palpated easily.

2. A sharply localized region of tenderness is present at the junction of the anterior pillar of the tonsillar fossa with the base of the tongue. This corresponds to the insertion of the styloglossus muscle and does not invade the musculature of the tongue.

3. Tenderness of the mylohyoid muscle may be present, but the tongue, except as noted previously, is free of tenderness.

4. Rarely the superior constrictors are involved in the myalgia, and when this involvement is present, a pain referred to the ear may be noted on swallowing, which suggests the diagnosis of glossopharyngeal neuralgia. This may represent an involvement of the glossopharyngeal nerve in the myalgia.

5. A localized region of tenderness is found over the ala of the thyroid cartilage, probably the oblique line of the thyroid cartilage, corresponding to the insertion of the sternothyroid muscle.

6. A localized region of tenderness is present along the lateral margins of the hyothyroid membrane, particularly at the superior cornu of the thyroid cartilage and the cornu of the hyoid bone.

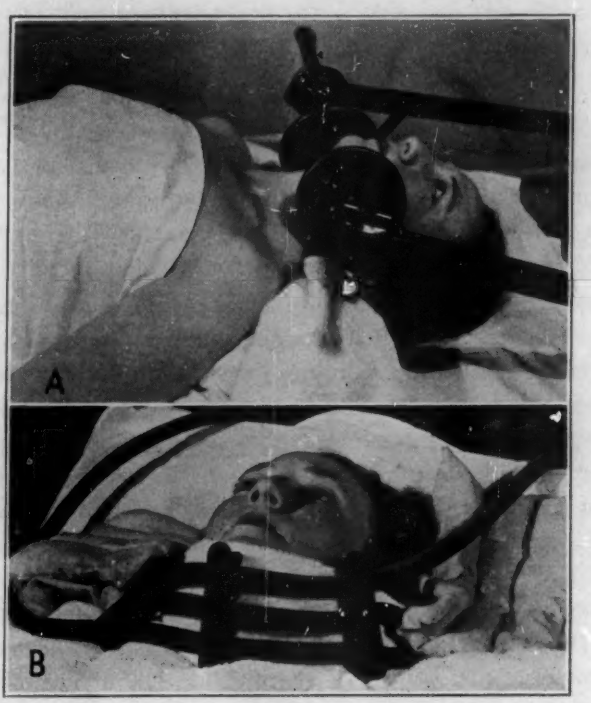
7. A localized region of tenderness is present on the posterior surface of the larynx, corresponding to the point of origin of the cricoarytenoid muscle.

In 1941, 18 patients have been seen who presented the foregoing symptom complex without any associated pains or tenderness elsewhere. This syndrome is seen frequently associated with myalgia of the head, however. It may be unilateral or bilateral. Instances have been observed in which involvement of the cricoarytenoid muscle was present on one side but involvement of the muscles having their origin from the styloid process was present on the other. An attack of myalgia of the pharynx is likely to be initiated by exposure of the anterior aspect of the neck to extreme cold, although a definite history of such exposure is not always obtainable. The syndrome may be present for a week to a month and disappear without leaving any residual sign or symptom. On the other hand, it may persist continuously for a year or longer.

DIFFERENTIAL DIAGNOSIS

Since it will be our purpose to attempt to demonstrate that myalgia is not associated with a so-called rheumatic process or with neuritis, the most important distinctions to be made in myalgia of the pharynx are that from primary fibrositis and occasionally that from glossopharyngeal neuralgia.

From Primary Fibrositis.—The characteristic symptoms of primary fibrositis are that, while it may appear and disappear, it tends to reappear



A, adjustment of air-spaced electrodes for the application of heat to the musculature of the neck and pharynx. *B*, application of a cable electrode.

in a different location. In myalgia recurrences are likely to be in the same location. In primary fibrositis subjective increase of stiffness from disuse, or "jelling," and relief from symptoms by moderate use of the involved muscles are prominent features. In myalgia, on the other hand, the symptoms are not increased by disuse nor are they relieved by moderate use of the involved muscles.

In primary fibrositis the administration of acetylsalicylic acid in sufficient doses gives complete or marked relief from the symptoms, while in

myalgia little or no relief can be obtained from even excessive doses of this drug.

From Glossopharyngeal Neuralgia.—In glossopharyngeal neuralgia anesthetization of the mucosa of the pharynx will give complete relief from the referred pain on swallowing, while in myalgia involving the superior constrictors no relief from the pain is afforded by anesthetization of the surface mucosa.

OBSERVATIONS IN THE LABORATORY SUGGESTING A MECHANISM OF PRODUCTION OF MYALGIA

In the past, observers have been inclined to attribute to myalgia a "rheumatic" origin or have expressed the belief that it was secondary to arthritis of one or another type. Recent work in the laboratory strongly suggests that myalgia is a disorder of function probably owing to sensitization to a chemical agent, which is probably histamine, and that restitution to a normal condition without secondary alteration of tissue takes place on disappearance of the syndrome.

In speaking of the localization of somatic pain, Lewis [2] stated that it was assumed too generally that somatic pain locates the seat of mischief. He expressed the belief that it locates the seat of mischief with great precision when this is the skin or an exposed mucous membrane but that there is much less evidence that it does so when deeper somatic structures, such as subcutaneous tissue or muscle, are concerned. Lewis noted that pain from the skin was well localized and that, while there were many variations in intensity, there was no variation in the quality of pain originating in the skin. The quality is the same however the pain is produced, whether from stimulation of nerve endings or of muscle fibers in the skin. This type of pain usually is described as burning. When convinced that pain derived from skin had a constant quality or tone, Lewis [2] tested muscle to see if pain originating from it possessed similar constancy. He injected isotonic acid solution (p_H 5 to 6.5) through a fine needle into a dorsal muscle of the forearm. On comparing the resulting pain with that produced simultaneously in the symmetric muscle by working this under ischemic conditions, Lewis found that the two pains were indistinguishable. He subsequently used hypertonic solutions (4 to 5 per cent) of sodium chloride or alternately a 0.5 per cent solution of potassium chloride. These injections gave identical pain. Lewis stated that the pain was disagreeable and continuous; its quality was indescribable, but the pain was distinctive. It was impossible to confuse pain from skin and pain from muscle.

Lewis [2] expressed the belief that the difference between the quality of cutaneous pain and the quality of deep pain is so clear, the quality of the former so exclusive to skin and mucous membrane and that of the latter to deep structures, that it is possibly an error to class both together under the one word "pain." It is a matter of usage; these two sensations have not been shown to possess the common properties which the use of a single term would imply. If the conclusion is correct that the system of fibers subserving cutaneous pain passes to an appropriate and exclusive part of the sensorium which determines this particular sensation, one is brought to consider whether fibers subserving pain derived more deeply do not connect to a separate part of the sensorium. Painful stimulation of the skin is well known to awaken quick protective reflexes, while painful stimulation of deeper structures does not appear to possess this association. While painful sensations derived from the human skin are associated with brisk movements, a rise of pulse rate and a sense of invigoration, those derived from deeper structures often are associated with quiescence, slowing of the pulse, a fall of blood pressure and nausea, the last phenomenon being responsible for the common designation "sickening," which is applied to this but never to cutaneous pain.

Lewis,[2] having noticed that pain arising from muscle might be referred to a distance, suggested that a systematic examination of pain provoked from the accessible muscles of normal subjects be made by means of the injection method. This was done by Kellgren,[3] who concluded that the diffuse pain from a given muscle always is distributed within certain regions. The distribution of the diffuse pain from muscle appeared to be in a segmental pattern. This pattern differed from that of the segmental innervation of the skin. Kellgren suggested two points which seemed to throw light on the mechanism of this referred pain. First, the pain does not appear to be referred to areas of skin but rather to deep structures; and second, pain arising from muscle appears to be confused by the subject with pain arising directly from these other deep structures. This suggests the possibility that the impulses responsible for pain from muscle and from the other deep structures may follow a final common path in the central nervous system. That referred pain from muscle is always a diffuse pain might be explained on the hypothesis that the pain fibers from muscle have diffuse instead of accurate synaptic connection with the central nervous system.

2. Lewis, T.: Suggestions Relating to the Study of Somatic Pain, Brit. M. J. **1**:321-325 (Feb. 12) 1938.

3. Kellgren, J. H.: Observations on Referred Pain Arising from Muscle, Clin. Sc. **3**:175-190 (April 28) 1938.

Kellgren [4] further reported investigation, from the point of view of his previous paper, of a number of patients who had "myalgia." The distribution of the pain was noted as accurately as possible, and experience of the distribution of pain provoked from normal muscles guided him to the muscles from which spontaneous pain might have arisen. Such muscles presented tender spots on palpation. Pressure on these spots sometimes reproduced the patient's pain, but a method more often successful was the injection of sterile saline solution into the tender muscle. When these acutely tender spots were not too extensive, they were infiltrated with 1 per cent solution of procaine hydrochloride. This infiltration often produced relief of the symptoms and signs and sometimes abolished them completely. In instances in which infiltration of procaine hydrochloride abolished the symptoms, this effect often outlasted the actual anesthesia and was more or less permanent.

On normal subjects, Lewis, Pickering and Rothschild [5] were able to demonstrate that there is no evidence that when a normal muscle to which blood flow has been stopped is exercised, the blood vessels enter a state of spasm; on the contrary, the vessels that supply the exercised muscles are dilated when the pain is acute. They also showed that the pain of muscular exercise in the absence of blood supply is determined by a stable chemical or physicochemical stimulus developed in the muscle during its exercise. The pain is not the direct result of lack of oxygen but is due to failure of oxygen to combine in a restorative physicochemical reaction.

Ray and Wolff [6] stated that pain was produced by any method that produced distention of an arterial wall. When distention was produced at two separate sites, the resulting pain seemed to be more widespread than the sum of the pains resulting from two separate distentions and was associated with a feeling of nausea, or sickness. Constriction of the lumen was not painful. The repeated application of epinephrine to exposed arteries otherwise sensitive to pain caused vigorous constriction of the arteries but no pain. The introduction of procaine hydrochloride into the adventitia of an artery immediately produced anesthesia to all stimuli a few centimeters distal to the point of injection. The implication is that the sensory nerve supply originates near the arteries and travels along with them.

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4. Kellgren, J. H.: A Preliminary Account of Referred Pains Arising from Muscle, Brit. M. J. **1**:325-327 (Feb. 12) 1938.

5. Lewis, T.; Pickering, G. W., and Rothschild, P.: Observations upon Muscular Pain in Intermittent Claudication, Heart **15**:359-383 (July 27) 1931.

6. Ray, B. S., and Wolff, H. G.: Experimental Studies on Headache: Pain-Sensitive Structures of the Head and Their Significance in Headache, Arch. Surg. **41**:813-856 (Oct.) 1940.

Pickering and Hess [7] suggested that the headache produced by the intravenous injection of histamine would be explained completely if the headache were due to stretching of a sensitive structure lying close to the meningeal arteries; such stretching might result from swelling of the perivascular tissues or from widening of the meningeal arteries. While amyl nitrite will produce an expansion of the brain and a lowering of the blood pressure identical with that produced by histamine, it will not produce headache. It is known that histamine increases the permeability of the minute blood vessels. Amyl nitrite has not been shown to do this. An increase in vascular permeability might be a factor in producing headache if the pain were due to stretching of the perivascular structures from edema secondary to the increased permeability of the vessels.

Lewis and Hess [8] stated that skin which is injured sufficiently in any way enters sooner or later a "susceptible state." The "susceptible state" is the immediate result not of injury but of ensuing inflammation. It is caused by the liberation of some tissue substance which acts on pain nerve endings. When skin in the "susceptible state" is rubbed, pain appears as a delayed after-effect. This pain is caused by the further liberation of the same tissue substance, the concentration of which now rises to a level adequate to produce pain. The mechanism which produces the pain referred to here is quite distinct from the excitation of pain nerve endings during actual stimulation of the skin. A "susceptible state" in the skin may occur, in which physical agents, such as temperature change, acting on the skin liberate a substance from the skin cells which plays on the endings of the pain nerves. The evidence suggests that the release of this substance is especially likely to occur in association with a state of inflammation. Lewis and Hess expressed the belief that there was considerable probability that such a substance was liberated in subcutaneous as well as cutaneous tissues.

In regard to pain, Lewis and Hess [8] stated that "smooth pain" can be regarded as arising not directly from the primary stimulus but through a relatively stable intermediate process. Such pain has a more delayed onset, rising gradually to its summit without any appreciable change and declining very slowly. The first pain of this class to be recognized was that arising in somatic muscle working without blood supply. Lewis [9] expressed the opinion that the pain arising in the skin from the release

7. Pickering, G. W., and Hess, W.: Observations on the Mechanism of Headache Produced by Histamine, Clin. Sc. **1**:77-101 (July 21) 1933.

8. Lewis, T., and Hess, W.: Pain Derived from the Skin and the Mechanism of Its Production, Clin. Sc. **1**:39-61 (July 21) 1933.

9. Lewis, T.: Clinical Observations and Experiments Relating to Burning Pain in the Extremities, and to So-Called "Erythromelalgia" in Particular, Clin. Sc. **1**:175-211 (Dec. 21) 1933.

of a substance, probably histamine, and the pain arising in muscle working under ischemic conditions are of the same general class. In his opinion the pain of angina pectoris has almost certainly a similar origin.

Horton and Roth [10] demonstrated that the substance liberated from sensitized skin on exposure to cold is histamine.

Schneider [11] stated that in the respiration of muscle the final interchange is between the active contractile substance of the muscle cell and the capillary wall by way of the sarcolemma, through which "chemical respiration" takes place. In the hypothetic case of vasodilation produced by histamine, with the chemical changes produced in the wall of the blood vessel which are undoubtedly present, it is possible and even probable that lack of oxygen in the biologically functioning muscle substance could be produced. This would result in the local collection of the "pain-producing substance" suggested by Lewis and his co-workers,[5] which Horton and Roth [10] have suggested is histamine. Schneider [11] also stated that the postural, or "red," muscles are less irritable, give a greater contraction in tetanus and can maintain the contraction for a longer period than can the more highly specialized "white" muscles. Schneider stated that this difference resides in the greater viscosity of the less specialized muscle. He also showed that fluid collects in muscles during activity and afterward may remain in them for some hours. In this condition a muscle is shorter, of greater girth and stiffer than normal muscles. Thus, in the postural muscles, which are normally in a state of tension for long periods, "myalgia" would have a greater tendency to develop.

Comment.—It is evident from the foregoing review of the literature that a tendency to sensitization of certain muscle cells to a chemical agent, probably histamine, could produce the symptom complex known as myalgia. Lewis pointed out that histamine produces not only a dilation of the blood vessels but an edema of their walls sufficient to cause interference with the fibers carrying pain sensation and running in close proximity to the vessel walls. This pain is of a distinctive sort and tends to be referred in a distribution which does not correspond to the spinal segmental cutaneous distribution but which may be vascular. The pain of myalgia is of this character. Ray and Wolff demonstrated that this pain, being, as suggested, of a vascular nature, must be on the basis of a vasodilation. The statement of Pickering and Hess [7] that physical trauma, such as that caused by massage, frees a substance,

10. Horton, B. T., and Roth, G. M.: Collapse While Swimming: The Most Dangerous Consequence of Hypersensitiveness to Cold, Proc. Staff Meet., Mayo Clin. **12**:7-10 (Jan. 6) 1937.

11. Schneider, E. C.: Physiology of Muscular Activity, ed. 2, Philadelphia, W. B. Saunders Company, 1939.

probably histamine, from the cells suggests that one mechanism by which massage and heat may produce their good results may be a process of desensitization from the slow release of histamine. Kellgren has demonstrated that myalgia is not a condition localized to the head but is merely more prominent subjectively when there because of its location and the reflex symptoms produced.

TREATMENT

If any one of the various theoretic concepts pointed out as to the cause of myalgia and its relation to pain is correct, then the use of certain physical therapeutic measures should be of benefit. Clinical experience is beginning to verify this, since the use of various forms of heat and a specific type of massage have been effective in bringing about relief of this condition. Some of the physiologic effects of heat which may be of benefit in relation to myalgia are: 1. There is increased circulation in the area to which heat is applied. 2. Changes occur in the p_H of the blood and probably also in that of the tissues. With local heating there is increased acidity, and with prolonged heating there is a change to alkalinity. 3. Heat is thought to produce some intracellular substances which decrease the resistance of the capillary wall to rupture. 4. Heat also increases the permeability of the capillary walls to certain cellular and fluid constituents of the blood which may be of importance in the treatment of myalgia if the cause is chemical. 5. Flow of lymph is increased, and this aids in decreasing edema. 6. Heat relaxes muscles and aids in the treatment of tension states which are present so often in this condition. 7. Heat has a certain amount of sedative effect on the sensory nerve endings and tends to lessen pain and irritation of nerves.

There are several sources of heat which may be used effectively. Among these are the luminous heat lamps, which produce the short infra-red rays—the most penetrative of the infra-red spectrum. The wattage of these lamps varies from 250 to 1,000. In cases of myalgia of the pharynx the heat is applied over the area which may be involved. One lamp has now been developed which has a black glass filter that prevents glare when it is in use over the face of the patient but at the same time allows transmission of the short infra-red rays. We consider that this form of heat should be applied for about twenty to forty minutes daily over a period of several weeks. The lamp should be used at a distance which will produce a comfortable warmth without a sensation of burning.

The high frequency currents, particularly short wave diathermy, are effective in producing a more penetrative heat than any of the other sources of heat used in physical therapy. They may be applied by means

of various technics. 1. The air-spaced electrode may be used. This technic permits adjustment of the electrodes over the neck at various angles, bilateral or unilateral, by means of adjustable arms (figure, *A*). If the involvement is on one side, a small air-spaced electrode can be adjusted to within 1 to 1½ inches (2.5 to 3.8 cm.) of the tender area and the large electrode can be placed over the shoulder or chest of the opposite side. 2. The pad technic may be used by placing one of the pads, preferably a small one, over the involved area and a larger pad beneath the shoulder. Or, if the involvement is bilateral, electrodes of equal size may be used on the two sides of the neck. The air spacing necessary when pads are used is provided by means of terry cloth or felt pads. 3. The electromagnetic induction cable technic may be used. In this case the cable may be draped to one side of the neck, or both sides of the neck if the involvement is bilateral, after sufficient air space has been provided by means of terry cloth padding or felt pads. This may be done by having the patient lie on the back with the head somewhat extended and placing two or three loops of the coil across the neck (figure, *B*), or it may be done by having the patient lie in the prone position over a pillow and directly on the air-spaced induction cable. The short wave diathermy treatments are given for about the same length of time as are the applications of the luminous heat lamps, namely, thirty to forty minutes.

If the foregoing apparatus is not at hand, hot towels, hot packs or paraffin may be used. The technic used in applying hot packs is to wring towels out of hot water, place them on the neck and cover them with rubber sheeting or dry towel. The packs should be changed about every five or ten minutes. Hot melted paraffin may be painted over the areas which are involved until a thickness of ⅛ to ¼ inch (0.3 to 0.6 cm.) is reached. The coating of paraffin is left in place for about thirty minutes. Sometimes it is essential for the patient to use this type of treatment in his home, when he does not have access to electricity.

Massage seems to be the most effective single agent in the treatment of myalgia of the pharynx. Used alone it is more effective than heat used alone, and a firm, heavy friction type of massage is more effective than stroking or kneading types.

The technic used in the treatment of the patients reported in this discussion consisted in applying mild forms of heat by means of luminous infra-red ray sources, or occasionally, when it was desired to treat more deeply involved areas, using short wave diathermy, for periods of thirty minutes. After this, a stroking type of massage was used at the beginning and then pressure progressively increased to a firm friction type of massage over the tender areas. The patient should be in a supine position, with the head sufficiently extended over a pillow or folded towel

to eliminate the folds of the neck. Care should be taken that the head is not extended to the point at which there is tension of the involved muscles. Stroking massage is done from the chin toward the sternum. Light to moderate pressure is used at first. After this, the tender areas over the various muscles of the neck in which the myalgia occurs can be singled out. These tender areas are massaged by use of a firm frictional circular movement, with the tips of two or three fingers. Pressure may be increased as the massage proceeds. This type of frictional massage is done from three to five minutes and is followed again by a stroking massage of the entire anterior portion of the neck. The type of massage allows treatment of almost any of the areas which may be involved around the larynx and the points of insertion of the various muscles of the pharynx and hyoid bone. Usually the total time of application of massage is ten minutes.

The friction type of massage is used to the extent of the patient's tolerance. Intervening periods of sedative, or stroking and kneading, massage are used when the friction massage becomes too painful. The friction massage is always followed by sedative massage. In the acute stage of myalgia a very strenuous firm massage cannot always be used; it is better to start with a milder type of sedative massage at the first treatment and gradually to increase the pressure of the stroking massage and then to administer friction massage during the second or third treatment. At times the very firm friction massage may cause the tender areas to become much more tender, although they usually are less painful after the heavy type of massage. Occasionally these areas become intensely painful after massage. In such instances the firmness of the massage may be lessened for one or two treatments and then the pressure may be progressively increased again.

Knowledge of the number of treatments necessary to relieve this condition is somewhat indefinite at the present time. Usually a period of one to three or four weeks of daily treatment will effect considerable relief or complete remission of the condition. How long these remissions will last cannot be stated; but usually if the myalgia recurs, the patient can control the discomfort readily by resuming treatment, either by professional means or at home.

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ACUTE MASTOIDITIS IN DIABETES MELLITUS

AN ANALYSIS OF FORTY-NINE CASES WITH A REPORT OF OBSERVATIONS AT NECROPSY IN ELEVEN

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The belief that there is impairment of immunity to infection in the tissues of the diabetic patient has been widely held for many years. In the field of otology this concept finds expression in the doctrine that in the presence of diabetes infections in the mastoid process are associated with relatively early and extensive bone destruction and are particularly prone to intracranial complications. Recently, however, exception has been taken to this view,[1] and it has been asserted that with the present improved therapy in diabetes, with especial reference to the use of insulin and a dietary regimen, the course of mastoiditis in the diabetic patient does not differ essentially from that in one who does not have diabetes. In an attempt to clarify this matter and to obtain, if possible, a better understanding of the influence of diabetes on the clinical manifestations, pathologic changes and treatment of mastoiditis, we have reviewed all cases of infection of the mastoid occurring in persons with diabetes admitted to the Mount Sinai Hospital within a period of fifteen years.

During the fifteen year period (1927 to 1941) there were admitted 49 patients with diabetes associated with acute mastoiditis. In 4 of these the disease in the mastoid was bilateral. Mastoidectomy was performed on 44. An analysis of the data on the 49 patients follows:

SEX

There were 29 females and 20 males. This preponderance of females is contrary to our experience with acute mastoiditis in persons who do not have diabetes, among whom males are somewhat more numerous than females. This incidence, however, approaches that of diabetes in the general population, in which the disease occurs more frequently in females.

From the Otologic Service and the Laboratories of the Mount Sinai Hospital.

1. Kecht, B., and Dibold, H.: Diabetes und Eiterungen im Mittelohr, Nasennebenhöhlen und Rachen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:288, 1936. Dibold, H., and Huber, K.: Wundheilung bei Diabetes, Fortschr. d. Therap. **14**:113, 1938. Majer, E. H.: Otitis bei Diabetes, Monatschr. f. Ohrenh. **73**:305, 1939.

AGE

Forty-two of the 49 patients were over 40 years of age. So large a proportion of older patients is not observed among nondiabetic persons with acute mastoiditis. However, diabetes, as is well known,[2] is most prevalent in later life, and it is to be expected that this preponderance would be reflected in the series herein presented.

ORGANISM

Pneumococcus type III was isolated in 21 cases, or 43 per cent, and Streptococcus haemolyticus beta in 15 cases, or 31 per cent. The pathogenic organisms less frequently isolated were Pneumococcus types II, IV and VII, Staphylococcus aureus and Streptococcus viridans. In 5 instances the organism was not identified. The preponderance of the type III pneumococcus over Str. haemolyticus is noteworthy. This was observed also by Barth.[3] It must not be construed from these figures that the type III pneumococcus has a particular affinity for persons with diabetes. A review of 1,000 consecutive cases of acute mastoiditis, including those of patients of all ages, encountered at the Mount Sinai Hospital, disclosed an incidence of 9 per cent for the type III pneumococcus and of 85 per cent for the streptococcus. However, in the age group past 40, which more nearly approximates that of the patients with diabetes, the incidence of the type III pneumococcus rose to 39 per cent. This incidence of 39 per cent corresponds very closely to the figure 43 per cent pertaining to the series under discussion. This would indicate that the age of the patient rather than the peculiarities of diabetes per se is the important factor in determining the character of the invading organism, particularly the type III pneumococcus.

SEVERITY OF INFECTION

We have learned from clinical experience that the most opportune time for surgical intervention in acute mastoiditis is that in which a sufficient period has elapsed to allow localization of the infection and beginning resorption of the intercellular bony septums. This process of localization and resorption usually requires twelve to fourteen days from the onset of the middle ear infection. In 12 of the 49 patients in this series, however, it was found that this period was definitely shortened (average duration nine days). In 6 cases extensive destruction was encountered at operation, while in the remaining 6 the changes

2. Health Education to Control Diabetes, Quart Bull., City New York Dept. Health **8**:77 (Nov.) 1940.

3. Barth, H.: Otitis media acuta und Diabetes, Klin. Wchnschr. **16**:198 (Feb. 6) 1937.

were of a more moderate degree. These cases are too few to permit drawing conclusions. Furthermore, it is well known that early destruction of bone is occasionally encountered in nondiabetic patients. Nevertheless, the impression gained is that bone destruction takes place at an earlier date in the diabetic than in the nondiabetic patient.

With regard to the extent of disease throughout the temporal bone in the diabetic patient it can be stated that in our series extension beyond the confines of the mastoid bone was encountered at operation or post mortem in 20 instances (41 per cent). This strikingly high incidence of complications is more than twice that observed in nondiabetic patients (18 per cent). It is almost twice that (22 per cent) encountered in nondiabetic patients of the age group past 40, which more nearly corresponds to the age group in which diabetes is most prevalent. From these figures it may be deduced that the extent of destruction throughout the temporal bone in acute infection of the mastoid is decidedly greater in the diabetic than in the nondiabetic patient.

It must not, however, be inferred from the foregoing observations that this destructive process is due solely to decreased resistance of the tissues to infection. Further analysis discloses the fact that in 17 of the 20 cases with intracranial complications the infectious process had been permitted to progress for a period of more than two weeks before the patient was admitted to the hospital and before mastoidectomy could be performed. In 14 of these the duration of the otitic infection was as long as four weeks or more. The failure to diagnose the condition early and to exenterate the suppurative process in the mastoid bone of the diabetic patient must be considered an added factor contributing toward the relatively high incidence of intracranial complications.

INFECTIONS OF THE SOFT TISSUES

An observation of considerable clinical importance in our present series was the frequent occurrence of postoperative erysipelas and other infections of the soft tissues of the neck and scalp. Infections of this nature were noted in 12, or 27 per cent, of the patients operated on. In 3 patients the severity was such as ultimately to bring about a fatal issue. This incidence of infection of the soft tissues is extremely high compared with that of 2 per cent for nondiabetic patients. Moreover, the lesions are usually not nearly so severe in the nondiabetic as in the diabetic patients. In 9 of the 12 cases under discussion Str. haemolyticus was recovered. This seems to indicate that the resistance of the soft tissues to streptococcic infection is greatly diminished in the presence of diabetes.

Furthermore, it is of significance that these complications involving the soft tissues occurred during the period of hospitalization, while the

patients were under careful supervision, including, e. g., examination of the urine, determination of the sugar and carbon dioxide of the blood and regulation of the diet and insulin intake. It is thus strongly suggestive that the impairment of resistance to infection occurs not only in the tissues of uncontrolled diabetic patients, as is generally recognized,[4] but also in those of patients who are treated adequately according to currently accepted methods.

RELATION OF THE SEVERITY OF OTITIC INFECTION TO THE SEVERITY OF DIABETES

In analyzing our series of cases it was deemed advisable to determine if possible the effect of the severity of the diabetes on the otitic infection. The cases of diabetes were accordingly divided into a mild, a moderately severe and a severe group by employing as an arbitrary criterion the quantity of insulin required daily to maintain the urine sugar free in the period prior to the onset of the otitis. The amounts of insulin required for these groups were as follows: none for the mild, up to 35 units for the moderate and 35 units or more for the severe group. There were in all thus classified 21 cases of mild, 17 of moderate and 11 of severe diabetes. The incidence of intracranial complications in the respective groups was as follows: 52 per cent in the mild, 42 per cent in the moderate and 27 per cent in the severe. The incidence of postoperative complications involving the soft tissues was, respectively, 33 per cent, 27 per cent and 18 per cent.

We are aware that in an arbitrary classification of this kind there is considerable overlapping and that many other factors enter into consideration, e. g., the nature of the organism, the age of the patient, the duration of the illness; nevertheless, the foregoing figures indicate that there is no direct correlation between the severity of the diabetes and the incidence of intracranial complications or postoperative complications involving the soft tissues.

PATHOLOGIC CHANGES

Histologic examination was made of 11 temporal bones sectioned serially, each obtained from a diabetic patient who had died subsequent to a suppurative process in the middle ear. In addition, 3 temporal bones from 3 diabetic patients whose condition was uncomplicated by middle ear involvement were similarly examined. It should be stated at the outset that except for differences in severity the histologic changes in the

4. Wolf, O., quoted by Goldberg, S. A.: Gangrene and Infection of the Ear, Nose and Throat Complicating Diabetes Mellitus, Arch. Otolaryng. **32**:16 (July) 1940. Lassar, O., cited by Moolten, S. E.: Pulmonary Infection and Necrosis in Diabetes Mellitus, Arch. Int. Med. **66**:561 (Sept.) 1940.

various structures of the temporal bones from the diabetic patients with otitic infection were essentially no different from those seen in the temporal bones from the nondiabetic patients. The usual inflammatory cellular reaction within the mucous membrane of the tympanum and mastoid and adjacent pneumatic spaces and the usual resorption of the bone by osteoclasts and perforating vessels were observed. The presence of fat-laden cells in the inflammatory exudate and in the fixed tissues of the diabetic patients, while not characteristic for this disease, was not an infrequent occurrence, particularly in those with subacute infections

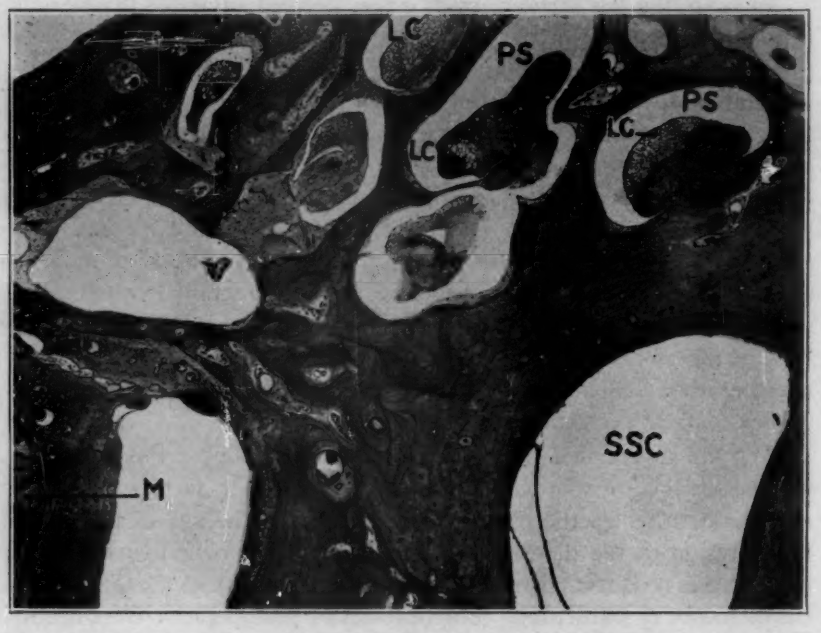

Fig. 1.—Vertical section through the petrosa. Lipoid cells can be seen within the inflammatory exudate throughout the pneumatic spaces of the petrosa. *LC* indicates lipoid cells; *M*, malleus; *PS*, pneumatic space, and *SSC*, superior semicircular canal.

(fig. 1). This may be attributed to hypercholesteremia and to faulty metabolism of fat in diabetes.

The rapidity with which the infection spread throughout the mastoid bone in 3 of the patients is a finding worthy of note. In each of these extensive destruction was observed to have occurred within one week after the onset of the otitis (fig. 2). We were struck also by the presence of a large number of intracranial complications (in 9 of 11 cases), which included labyrinthitis, petrositis, abscess of the brain, sinus thrombosis and meningitis, either alone or in combination (fig. 3).

Of further significance is the histologic observation in 5 of the 11 temporal bones sectioned of an acute inflammatory process in the skin lining the external auditory canal; the inflammatory changes consisted of cellular infiltration, hemorrhages and localized abscesses in the subepidermal tissue with ulcerations of the epidermis (fig. 4). In 3 cases the changes in the external canal were in all probability secondary to

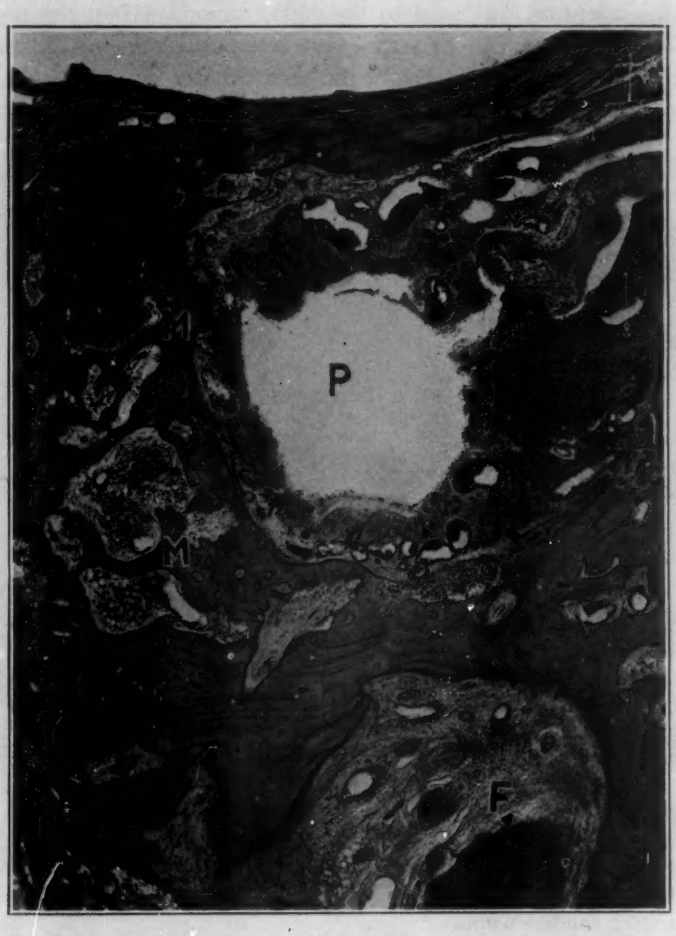

Fig. 2.—Section through the mastoid showing an extensive suppurative process in the pneumatic and marrow spaces, with osteoclastic resorption of bony margins. The patient had acute otitis media of five days' duration. *F* represents the facial nerve; *M*, marrow space, and *P* pneumatic space.

an inflammation within the middle ear and mastoid. They were not observed in any of the 3 cases in which diabetes was uncomplicated by middle ear suppuration.

Bearing in mind all other factors, one obtains from these observations under the microscope the impression that the tissues of the diabetic patient offer considerably less resistance to the invading organism than do those of the nondiabetic patient.

In view of the fact that arteriosclerosis is a frequent complication of diabetes, and since delayed healing of wounds has been ascribed to it, particular attention was paid to the blood vessels within the temporal bone. In general it can be stated that sclerosis of the small arterioles was observed more frequently in the series under discussion than it is usually observed in nondiabetic conditions, the age of the patient being

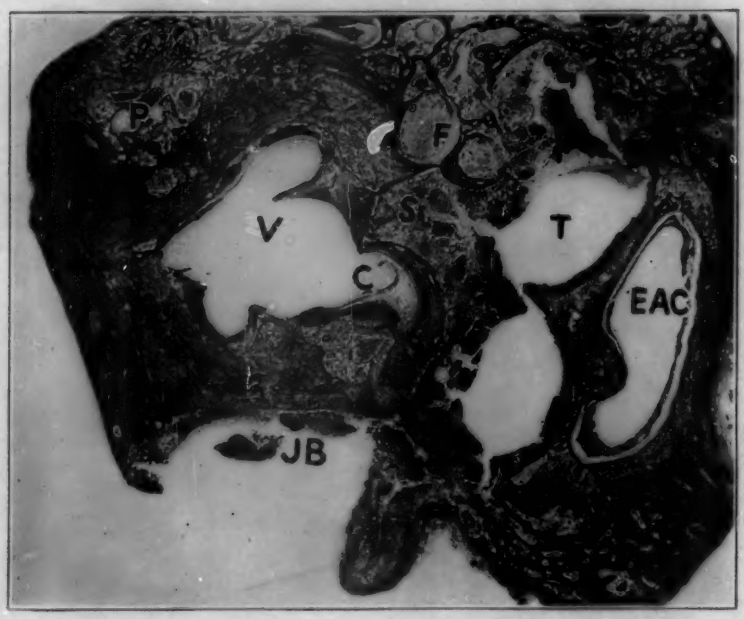

Fig. 3.—Vertical section through the tympanum and vestibule, showing evidence of a diffuse infectious process with production of labyrinthitis, petrositis and thrombosis of the bulb of the jugular vein. *C* represents the cochlea; *EAC*, the external auditory canal; *F*, the facial nerve; *JB*, the bulb of the jugular vein (thrombosed); *P*, the petrosa; *S*, the stapes; *T*, the tympanum, and *V*, the vestibule, with purulent exudate within.

taken into consideration. Sclerosis of the smaller vessels was also observed in one of the temporal bones of a diabetic patient in whom there was no otitic infection.

DIAGNOSIS

The importance of establishing a diagnosis of acute mastoiditis as soon as possible cannot be overstressed. A diabetic patient with acute suppuration of the middle ear should at all times be under close obser-

vation, so that the earliest signs of postauricular involvement may be detected. The relatively high incidence of intracranial complications in this series, in our opinion, is due partially to failure in diagnosing early the suppurative process in the mastoid bone. It has been frequently noted that in the presence of diabetes mastoiditis may supervene with a paucity of clinical signs and symptoms. This less well defined clinical picture of mastoiditis, however, should not be ascribed solely to the presence of diabetes but also to a number of other factors common particularly to older persons—e. g., changes in the vascular system, thickening of the bony cortex of the mastoid and frequent occurrence of

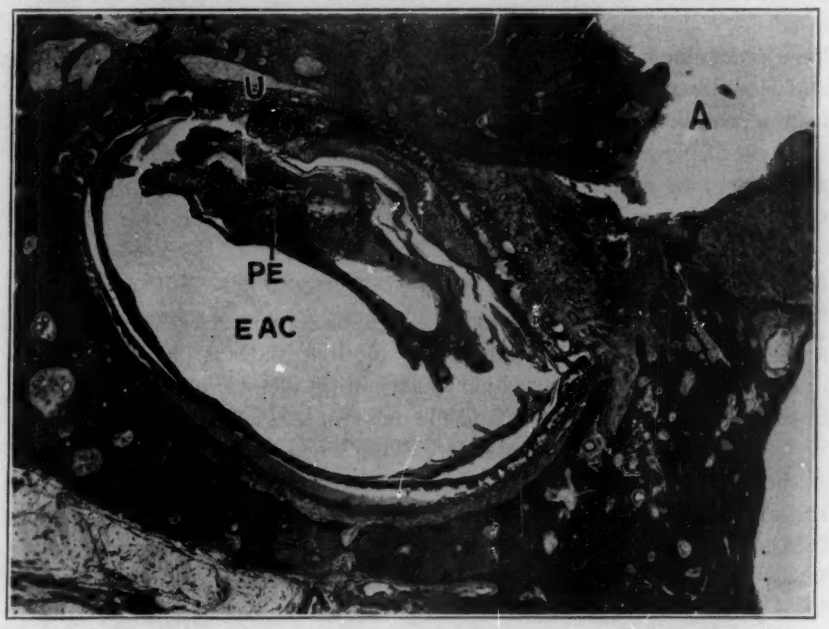

Fig. 4.—Photomicrograph of a section through the external auditory canal, showing marked inflammation of the integument with ulceration of the epidermis at *U*. Note the excessive amount of epidermal exfoliation. *A* indicates the mastoid antrum; *EAC*, the external auditory canal, and *PE*, purulent exudate with exfoliated epidermis.

the type III pneumococcus. The prolonged insidious course of mastoiditis, with few physical signs, in the presence of Pneumococcus type III is a well recognized clinical observation.[5]

5. Maybaum, J. L., and Druss, J. G.: Otitic Infections Due to the Pneumococcus Type III. Arch. Otolaryng. **30**:21 (July) 1939.

COMPLICATIONS

The complications can be divided into those involving the intracranial contents and those involving the soft tissues. Intracranial complications, as noted previously, occurred in 20 of the 49 patients. These included perisinal abscess, epidural abscess, petrositis, labyrinthitis, meningitis, brain abscess and thrombophlebitis of the lateral sinus. They occurred singly or in combination and were disclosed at operation or on postmortem examination.

Postoperative infection of the soft tissues occurred in 12 instances. In 6 of these it made itself manifest as erysipelas and in 6 as cellulitis of the subcutaneous tissues about the mastoid wound. The infection varied in severity and extent but in 4 instances was so pronounced as to cause huge phlegmons of the neck, scalp and chest wall and in 1 case necrosis and sloughing of the eyelids. Three deaths can be attributed directly to this complication.

It is also to be noted that inflammatory conditions of the external auditory canal and auricle were observed much more frequently in this series of patients than is usual in nondiabetic patients.

MORTALITY

There were 13 deaths, a mortality rate of 26 per cent. This is indeed a high death rate. To the important role that diabetes plays in its production must be added the other contributing factors, namely, the delay in diagnosing the mastoiditis and the delay in surgical intervention. The records disclose that 5 patients were dying when admitted to the hospital. If these are excluded, the mortality rate is lowered to 16 per cent. The causes of death were as follows: (1) intracranial complications in 7 instances (in 5, meningitis, in 1, abscess of the brain and in 1 thrombosis of the lateral sinus); (2) postoperative infection of the soft tissues in 3 instances; (3) nonotitic complications in 3 cases (anaphylactic shock in 1, fibrinopurulent pleuritis and pericarditis in a second and renal insufficiency with diabetic coma in a third).

TREATMENT

The following suggestions with regard to treatment are made. It is essential that treatment be directed toward the diabetes as well as toward the otitic infection. The diabetic condition, as is well known, is frequently aggravated by the intercurrent infection. Consequently, it is necessary to intensify the therapy accordingly. An attempt was made whenever possible to render the patient's urine free of sugar and acetone prior to surgical intervention. With the proper preliminary treatment, including the administration of saline and dextrose infusions and insulin,

the diabetic patients apparently tolerated the usual surgical procedures with no greater difficulty than did the nondiabetic. Difficulty in regulating the diabetic state was often due to the accompanying infection and was frequently obviated by subsequent removal of the infection. Protamine zinc insulin, as well as ordinary insulin, has been used during the past few years.

It is particularly important that a diagnosis of the condition calling for surgical intervention be made as soon as possible. Once the diagnosis is established and proper preliminary medication has been administered, operative intervention should be carried out without delay. A study of our cases indicates, as previously mentioned, that the high incidence of intracranial complications is, among other factors, definitely related to delay in diagnosing the condition requiring surgical treatment and to delay in performing the necessary operation.

With regard to anesthesia, it is noteworthy that the patients seemed to tolerate nitrous oxide–oxygen and ether satisfactorily, particularly when necessary precautionary measures against acidosis had been taken. Only on rare occasions is the state of diabetes of such gravity as to require the use of local anesthesia. The importance of complete exenteration of all cellular elements in the performance of mastoidectomy cannot be overemphasized. At the same time particular care must be exercised in avoiding undue trauma to the adjacent soft parts, e. g., in the removal of the tip of the mastoid, in retracting the skin and in suturing of the soft tissues. The frequent occurrence of secondary infection of the soft parts with its serious consequences in diabetic patients must always be borne in mind by the surgeon.

Sulfanilamide and its derivatives serve a most useful function in the therapy of otitic infections in diabetic as in nondiabetic patients. Their use is particularly indicated when there are intracranial complications or postoperative infection of the soft tissues. Although these drugs were not yet available for use in the great majority of cases in this series, nevertheless their value in combating intracranal or impending intracranial complications has been so amply demonstrated elsewhere as to necessitate no further comment here. They were employed, however, in 3 cases in which there was postoperative infection of the soft tissues, with excellent results in each.

On the other hand, we do not recommend the use of sulfanilamide and its derivatives in cases in which mastoiditis is suspected or overt. In a case in which mastoiditis is suspected, the picture may be obscured by the masking of signs and symptoms; in a case in which the condition is overt, with bone suppuration, surgical treatment cannot be replaced satisfactorily by the administration of drugs.

SUMMARY AND CONCLUSION

An analytic study is made of 49 cases of acute mastoiditis associated with diabetes, to determine if possible the effect of the diabetes on the clinical course, pathologic changes, treatment and outcome of the aural disease.

The significant observations are as follows: An unusually high incidence of intracranial complications of all types was encountered (41 per cent). The incidence of postoperative erysipelas and of infections of the soft tissues about the mastoid wound was likewise high (24 per cent). These infections of the soft tissues were not infrequently very severe and occurred despite vigorous treatment of the diabetes. A correlation between the severity of the diabetes and the incidence of intracranial complications or of postoperative infections of the soft tissues could not be made.

Histologic examination of 11 temporal bones corroborated some of our clinical observations. In addition, it disclosed an increase in the amount of sclerosis of the smaller blood vessels and of deposition of lipoids within the inflammatory exudate of the temporal bone.

The following suggestions with regard to therapy are made: 1. Treatment should be directed toward the diabetes as well as toward the otitic infection. 2. It is important to establish the diagnosis of mastoiditis early and to avoid undue delay in surgical intervention. 3. It is important to exenterate completely all diseased tissue at operation. 4. Undue trauma to the external auditory canal in cleansing and to the soft parts about the mastoid wound at operation should be avoided. 5. The administration of sulfanilamide or one of its derivatives is strongly indicated in the presence of impending or apparent intracranial complications and in the presence of infections of the soft parts.

It is concluded that there occurs in the diabetic patient an impairment of resistance of the temporal bone and adjacent soft tissues to infection.

1040 Park Avenue.

40 West Eighty-Sixth Street.

A MODIFIED FENESTRATION TECHNIC

ANALYSIS OF RESULTS IN ONE HUNDRED AND FORTY-ONE CONSECUTIVE OPERATIONS

GEORGE E. SHAMBAUGH JR., M.D.
CHICAGO

For more than half a century, in fact, since the condition was first recognized as a pathologic and clinical entity, otologists have sought a method of alleviating the hearing impairment of otosclerosis by medical or surgical means. One method attempted by such eminent otologists as Passow, Bárány and Holmgren has been the surgical construction of a new labyrinthine window to take the place of the occluded oval window. Sourdille, using a complicated and prolonged three or four stage operative technic, appears to have been the first to secure lasting and significant hearing improvement in cases of otosclerosis. Lempert, in 1938, modified, simplified and combined Sourdille's series of operations into a single, one stage fenestration operation, which for the first time seemed to offer the possibility of producing a lasting improvement of hearing in cases of otosclerosis by means of a practical one stage operation. This work has been followed by an extraordinary and widespread growth of interest in the surgical treatment of otosclerosis. Much comment, some of it critical and unconstructive, has greeted the fenestration operation and has tended to obscure its true value and significance. However, sufficient time has elapsed to indicate, on the one hand, that Lempert's original fenestration technic yielded results which still left much to be desired but that, on the other hand, it produced in some cases significant hearing improvements that have been maintained without appreciable loss for as long as four years. It seems clear that Lempert's work constitutes a contribution, and an important and outstanding one, to the development of the surgical treatment of otosclerosis, but that it should be regarded not as the final word but rather as another step forward in the development of a safe, reliable and effective means of improving the hearing in this disease.

In the last analysis results will speak for themselves, and the evaluation of any surgical technic for improving hearing in cases of otosclerosis must be based on complete and accurate data on late as well as early results. It is my intention to present my results with a modified fenestration technic in sufficient detail so that they may speak for themselves. It is my hope that these results may indicate that my modifications are a further small step forward in the surgical treatment of otosclerosis.

The reason I adopted the modifications in the fenestration technic which I shall shortly describe was that my experiences with the original fenestration technic were disappointing. These experiences consisted of 15 fenestration operations done between July 1938 and January 1940

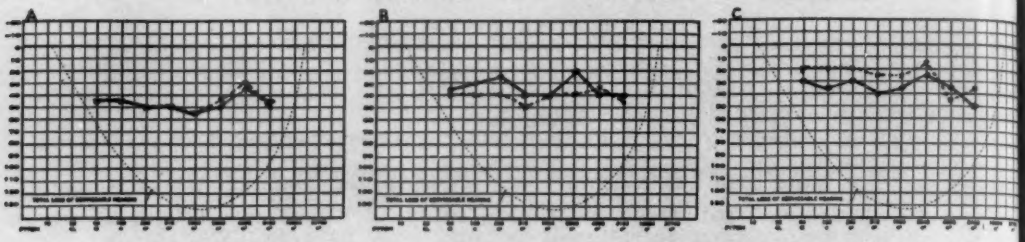

Fig. 1.—Audiograms for the only patient who experienced a satisfactory and lasting hearing improvement after the original fenestration technic. *A* is the preoperative audiogram; *B*, the audiogram taken four months after fenestration operation on the right ear, and *C*, the audiogram taken twenty-six months after operation on the right ear and fourteen months after operation on the left ear.

In these and the accompanying audiograms, the continuous line represents hearing in the right ear and the dotted line, that in the left ear.

Fig. 2.—Drawing to show technic of revision. The endaural incision is reopened and the epidermal lining of the operative cavity, freed by a posterior incision along the facial ridge and an anterosuperior incision, is elevated forward, exposing the bony horizontal semicircular canal and the incus. The irrigating attachment is placed on the self-retaining endaural retractor, and the binocular microscope is brought into position. The circle shows the region seen through the microscope.

on 13 patients with stapes fixation resulting from otosclerosis. In each instance Lempert's fenestration operation was accomplished correctly

as nearly as I could judge on the basis of previous observation of his work and his instruction in the technic on the cadaver. In no case was the tympanomeatal skin flap torn, the incus dislocated or the membranous labyrinth destroyed. Although an initial hearing improvement occurred in 13 of the 15 cases, a lasting improvement of more than 10 decibels for the conversational frequencies (512, 1024 and 2048) was secured

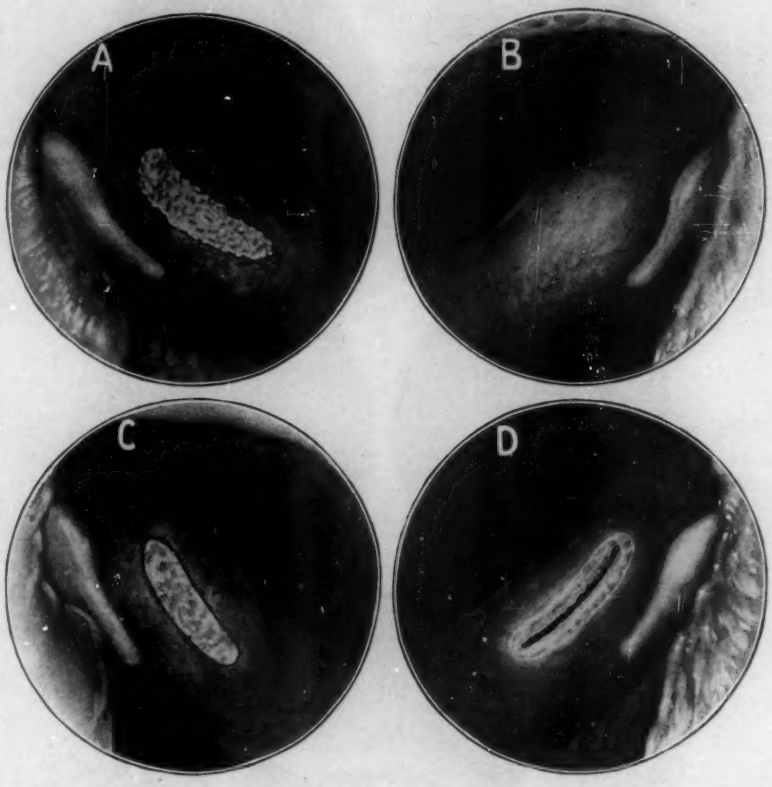

Fig. 3.—Revisions. *A* (case 1), fistula closed by a lid of whitish bone.

B (case 4), site of fistula obliterated by new bone.

C (case 5), fistula filled with a movable plug of granular bone, probably derived from bone dust.

D (case 7; right ear), fistula narrowed to a slit by new bone.

in only 4 instances, while in only 1 of these was improvement sufficiently great to be regarded as satisfactory (fig. 1 and tables 2 and 8).

Subsequently, in 10 of these first 15 cases revisions were made, affording an opportunity to inspect the fistula many months after fenestration and to speculate on the cause of failure. The appearances of these fistulas at the revisions are shown in figures 2 to 4. Several

types of gross pathologic changes causing closure of the fistula were observed (table 1).

In cases 1, 7 (both ears), 10 and 12 (figs. 3 *A*, 3 *D*, 4 *B* and 4 *D*) the surface of the bony horizontal semicircular canal, exposed by elevating the tympanomeatal flap, appeared exactly as at the conclusion

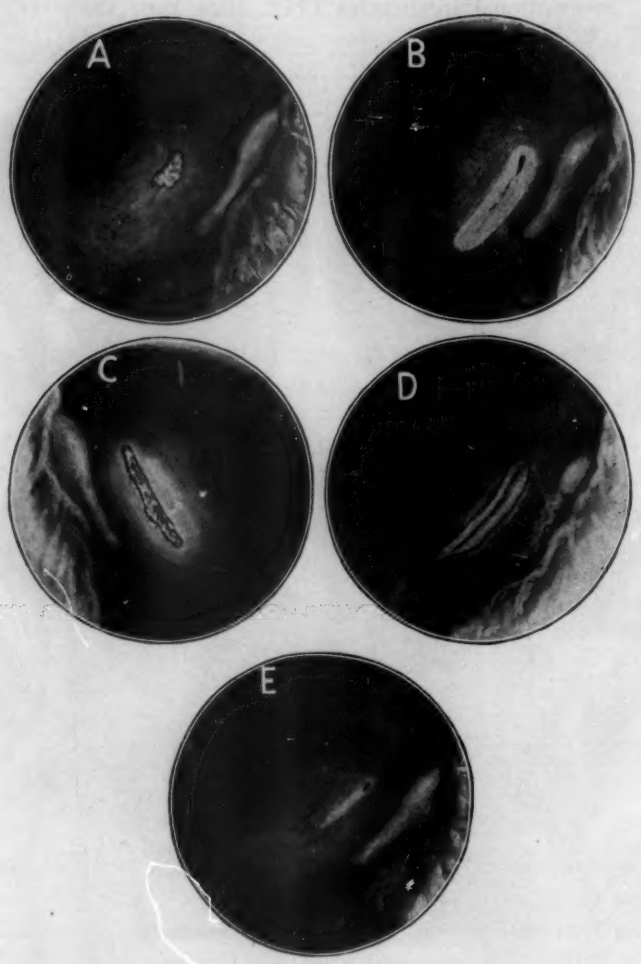

Fig. 4.—Revisions. *A*, fistula closed.

B (case 10), fistula partially closed.

C (case 11), fistula slightly narrowed and filled with a movable plug of granular bone dust.

D (case 12), fistula closed.

E (case 13), fistula partially closed.

of the previous operation, with no visible bone regeneration except for a sharply demarcated lid of whitish new bone covering the fistula and

TABLE 1.—*Summary of Data on Revisions After Original Lempert Operation*

Case No.	Ear	Hearing Result at Time of Revision: Time After Operation, Mo.	Hearing Result at Time of Revision: Fistula Test	Hearing Result at Time of Revision: Decibel Change for Speech	Observations at Revision	Technic *	Final (Most Recent) Hearing Result: Time After Operation, Mo.	Final (Most Recent) Hearing Result: Fistula Test	Final (Most Recent) Hearing Result: Decibel Change for Speech
1	L	18	—	+ 7	Surface of bony canal unchanged; fistula closed by plug of granular whitish bone (fig. 3*A*)	Thiersch graft	24	++++	+37
4	R	26	—	+ 2	Regeneration of bone on surface of bony canal obliterating the site of the fistula (fig. 3*B*)	Thiersch graft; irrigation and micro.	16	++++	+15
5	R	25	++++	—10	Surface of canal unchanged; movable plug of granular bone filling fistula (fig. 3*C*)	Irrigation; 2d fistula in superior canal	15	++++	+25
7	L	24	—	+15	Surface of canal unchanged; fistula closed by thin layer of white bone, distinct from labyrinthine capsule	Thiersch graft; irrigation and micro.	13	++++	+37
7	R	15	—	+12	Surface of canal unchanged; fistula narrowed to a slit by white bone, distinct from labyrinthine capsule (fig. 3*D*)	Thiersch graft; irrigation	19	++++	+24
9	R	12	—	—11	Bone regeneration on the surface of canal obliterating the site of the fistula except for a tiny area of whitish bone (fig. 4*A*)	Thiersch graft; irrigation	16	++++	+24
10	R	17	+	— 3	Surface of canal unchanged; fistula closed by thin layer of white bone, distinct from labyrinthine capsule except for tiny opening anteriorly (fig. 4*B*)	Irrigation and micro.	11	++++	+18
11	L	10	++++	+11	Bone regeneration on surface of canal narrowing fistula; movable plug of white bone in fistula (fig. 4*C*)	Thiersch graft; irrigation	15	++++	+20
12	R	22	—	+ 1	Surface of canal unchanged; fistula closed by very thin layer of whitish bone, distinct from labyrinthine capsule (fig. 4*D*)	Irrigation and micro. (novovalis)	4	++++	+24
13	R	4	+	+ 3	Bone regeneration on surface of canal partly obliterating fistula; whitish bone, distinct from rest of capsule, covering rest of the fistula except for a tiny opening anteriorly (fig. 4*E*)	Thiersch graft; irrigation	8	+	+ 1
13	R, 2d revision	9	+	+ 1	Thiersch graft gone; bone regeneration on surface of canal and layer of whitish bone narrowing fistula to slit; lumen of canal partly filled with fibrous tissue	Irrigation and micro.; 2d fistula made in posterior canal	12	+++	+18

* "Micro." indicates that use of the dissecting microscope was introduced into the operation.

distinct from the slightly ivory-colored bone of the labyrinthine capsule. The gross appearance suggested that this new bone was largely derived from the endosteum.

In case 4 (fig. 3 *B*), on the other hand, the surface of the bony horizontal semicircular canal was covered by new bone, which completely obliterated any evidence of the site of the fistula. The gross appearance suggested that the new bone was derived from the periosteal layer of the labyrinthine capsule.

In cases 5 and 11 (figs. 3 *C* and 4 *C*) an occluding but movable plug of whitish, granular-appearing bone filled the fistula, permitting a positive response to the fistula test but not allowing the transmission of sound impulses. The gross appearance here suggested that bone dust which had fallen into the perilymph space had organized to form the plug of new bone.

In case 7 (right ear), 10 and 13 (figs. 3 *D*, 4 *B* and 4 *E*) a tiny opening into the perilymph space was still present, permitting a positive reaction in the fistula test but being too small to allow the free transmission of sound impulses.

In case 13 at the second revision the lumen of the horizontal semicircular canal was observed to be occluded for part of its length by fibrous tissue.

To summarize, on 10 ears operated on by the original Lempert fenestration technic revision was carried out because of partial or complete loss of the hearing gain (in 3 and 6 ears, respectively) or failure to secure hearing improvement at any time (in 1 ear). At the time of revision a positive response was still obtained in the fistula test in 4 of the 10 ears—weak (1 plus) in 2 and very active and strong (4 plus) in the other 2.

At every revision failure of the first operation was found to be due to partial or complete closure of the fistula by new bone.

Because chips and fragments of bone had been shown to be of great importance in the formation of new bone, it seemed probable to me that incomplete removal of bone dust was a factor in the bony closure of at least some of these fistulas. For example, in case 5 I had seen bone dust created by the dental finishing burr accumulating in the perilymph space, but I had not known how to remove it without endangering the membranous labyrinth. Stimulated by the experience of a colleague whose best fenestration result followed accidental laceration of the dura of the middle fossa, with leakage of spinal fluid that kept the bony horizontal canal washed clean during creation of the fistula, I devised a simple irrigating attachment for a self-retaining endaural retractor (fig. 5). With this device, warm sterile physiologic solution of sodium chloride

flows by gravity through one silver tube into the anterior end of the operative cavity, while suction removes the saline solution from the posterior end of the cavity through the other tube. The flow of saline solution in no way interferes with the making of the fistula; rather, it greatly improves the visualization of the structures by keeping the field immaculately clean at all times. Every particle of bone dust is instantly washed away as fast as it is created by the finishing burr, preventing any from falling into the perilymph space or remaining around the incus or adhering to the bone or to the tympanomeatal skin flap.

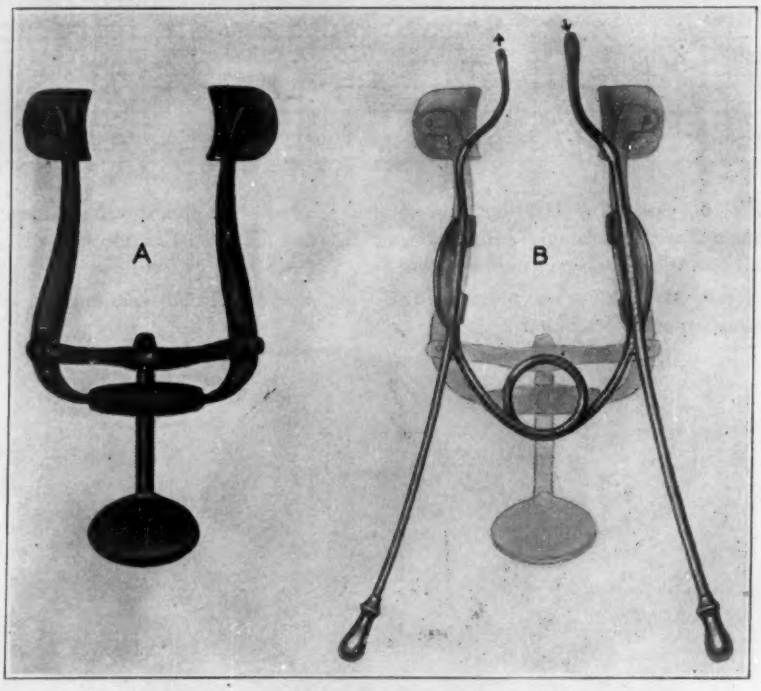

Fig. 5.—*A*, self-retaining retractor for the endaural approach, with gun metal finish to prevent glare.

B, irrigating apparatus which fits on to the self-retaining retractor, the smaller tube being for saline solution and the larger tube for suction. These tubes are of flexible silver and can be made to fit the walls of the operative cavity, so that they are out of the way.

These instruments are made by the American Optical Company, 10 South Wabash Avenue, Chicago.

The irrigating apparatus was first used in February 1940. For the first time I was able to visualize clearly the membranous labyrinth and to tease away the endosteum, with its adherent particles of bone, leaving

the membranous labyrinth floating in the widely opened perilymph space. The patient (fig. 6) has maintained a strongly positive response to the fistula test and a satisfactory hearing improvement, which after more than two years shows no sign of decreasing.

In July 1940, in doing a revision in case 16, the fistula was found to be closed partly by bone (fig. 7) and partly by dense fibrous tissue adherent to the membranous labyrinth. While attempting to pull the

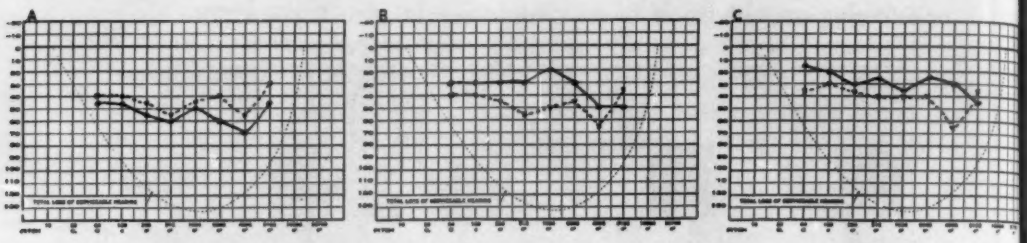

Fig. 6 (case 14).—Hearing gain of +29 decibels for the speech frequencies maintained with a strongly positive response in the fistula test in the first case in which operation was performed with the use of constant irrigation.

A was taken before operation; *B*, two months, and *C*, twenty-one months, after fenestration on the right ear.

Fig. 7 (case 16).—Revision. The fistula is partly closed; the lumen of the canal is filled with endosteal bone and dense fibrous tissue adherent to the membranous labyrinth. The membranous labyrinth has been accidentally avulsed.

fibrous tissue away from the membranous labyrinth, the latter was accidentally avulsed from the horizontal canal, leaving the lumen empty. An initial hearing improvement was soon lost, since the lumen of the canal became filled with fibrous tissue, as was observed at a second revision fifteen months later. This experience caused me to seek greater magnification than the binocular loupe affords in order to facilitate

manipulations involving the membranous labyrinth with a minimum of danger to this delicate structure. A Bausch and Lomb binocular dissecting microscope with a working distance of 4 inches (10 cm.) from the objectives and providing a magnification of seven was found to be satisfactory. Mounted on an adjustable arm and stand (fig. 8*A*) placed

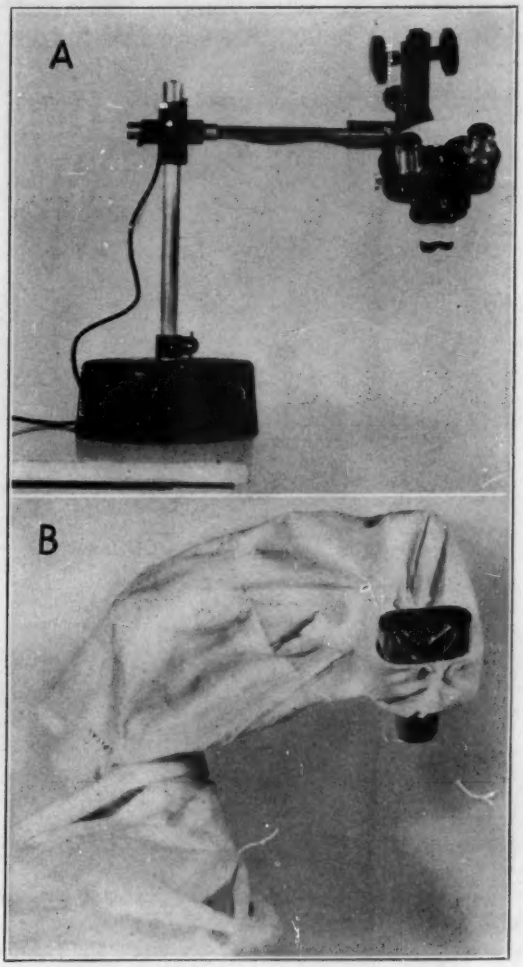

Fig. 8.—*A*, binocular dissecting microscope on adjustable arm. *B*, microscope covered with sterile sleeve and metal covers around the oculars and objectives.

on a small table with wheels and covered with a sterile sleeve (fig. 8*B*), the microscope is easily wheeled into position at the operating table (fig. 9). The use of this instrument has made the creation of the fistula much easier for me, particularly the important and difficult step of removing the endosteum, with its adherent particles of bone (fig. 10).

Statistically (tables 9 and 10), the use of the microscope has not made as much difference in the results as has the employment of irrigation, but because of the increased ease of making the fistula, I should not wish to do without it.

In addition to these two major modifications in the fenestration technic, I have adopted a few minor changes, which seem to me to be less important in the final result. These are the use of avertin with

Fig. 9.—Operating room setup, with the microscope and irrigating apparatus in use. Note the glass container for the warmed sterile physiologic solution of sodium chloride and the sterile sleeves covering the microscope and the dental engine arm and hand piece.

amylene hydrate to produce complete narcosis, the employment of very fine iridectomy forceps and scissors under the binocular magnifying loupe to trim down the tympanomeatal skin flap and to remove all adherent particles of bone; the use of two specially constructed, very tiny hooks to lift off the endosteum, and the employment of sulfanilamide, or sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine), powder in the

wound and of small doses of the drug by mouth after operation to minimize or eliminate the low grade postoperative infection that frequently becomes evident during the second or third week. With these modifications the time required for operation now ranges between one and one-half to three hours, averaging about two hours. The patient is usually allowed out of bed about the fourth day after operation and

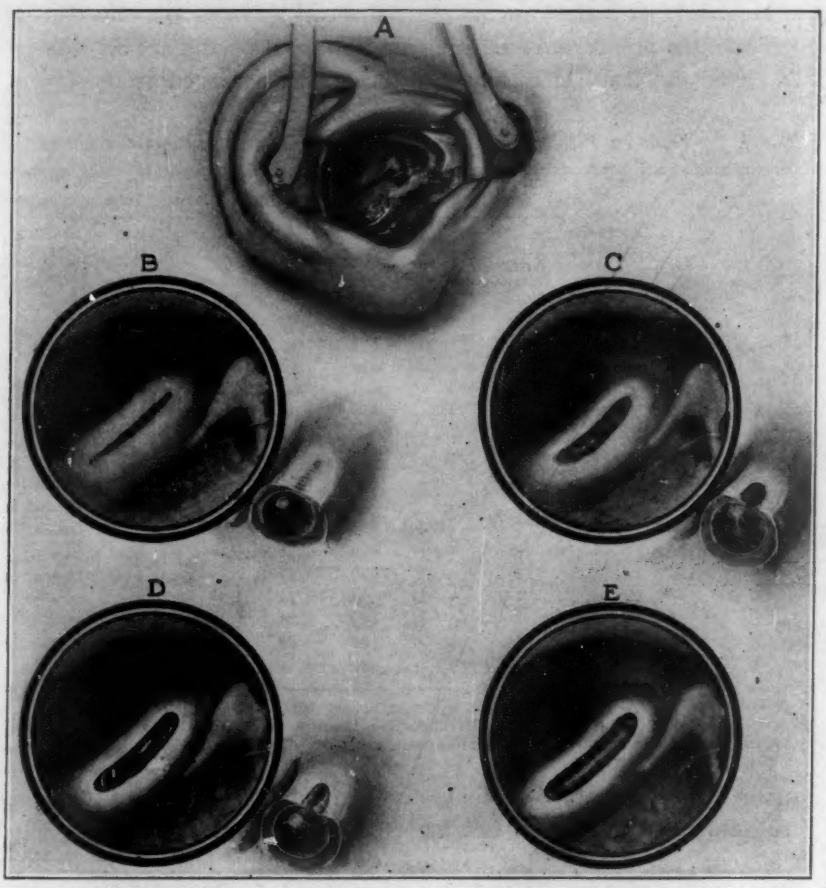

Fig. 10.—Technic of making the fenestra, using the microscope and constant irrigation.

A, bony horizontal semicircular canal defined, head of malleus removed and tympanomeatal skin flap prepared. The irrigating apparatus is now attached to the self-retaining retractor, and the microscope is brought into position.

B, lumen of horizontal semicircular canal beginning to show as a gray streak as the bone is slowly removed with the dental finishing burr.

C, last layer of endosteal bone covering the lumen of the canal fractures.

D, endosteum with adherent particles of endosteal bone still covering the fistula. The 14 karat, hard gold burnishing burr has polished the surface of the bony canal.

E, after the endosteum has been lifted off with tiny hooks, leaving the membranous labyrinth floating in the perilymphatic space. The fenestra is completed.

returns home on the seventh to the ninth day. Complete healing by epidermization of the operative cavity requires six to ten weeks in most cases, with eight weeks about the average.

PRESENTATION AND ANALYSIS OF RESULTS

In tables 2, 3 and 4 the important data on each fenestration operation are recorded, with the case number, the ear operated on, the date of operation, the preoperative hearing level for speech (the decibel loss for 512, 1024 and 2048 vibrations per second, as averaged from several,

TABLE 2.—*Data on Fifteen Ears Operated on by Original Lempert Technic* *

				Best Hearing Result			Final (Most Recent) Hearing Result			
Case No.	Ear	Date of Operation	Preoperative Hearing Level for Speech, Decibels	Time After Operation, Mo.	Fistula Test	Decibel Change for Speech	Time After Operation, Mo.	Fistula Test	Decibel Change for Speech	Decibel Change for Speech in Intact (Control) Ear
1	L	7/13/38	67	1	+	+19	18	—	+ 7	+ 1
2	R	7/20/38	53	2	++++	+16	33	++++	— 5	+ 1
3	R	7/27/38	52	24	—	— 5	36	—	—13	+ 4
4	R	8/17/38	64	1	+	+ 9	26	—	+ 2	+ 6
5	R	10/12/38	65	1	++++	— 3	23	++++	—10	+10
6	R	10/26/38	70	1	+	+20	26	—	0	+ 7
7	L	11/ 9/38	75	1	++++	+40	24	—	+15	Operation
7	R	1/10/39	72	2	+	+19	15	—	+12	Operation
8	R	7/13/39	52	1	++++	+20	26	++	+17	Operation
9	R	8/ 9/39	59	1	++++	+19	12	—	—11	— 2
1	R	8/16/39	61	1	+	+13	28	—	+ 4	Operation
10	R	11/ 2/39	60	1	++++	+27	15	+	— 3	+ 8
11	L	11/22/39	68	1	++++	+20	10	++++	+11	— 2
12	R	1/ 4/40	66	1	++	+19	22	—	+ 1	Operation
13	R	1/17/40	58	1	++++	+13	4	+	+ 3	+ 5

* The "hearing change for speech" is the average decibel loss or gain for speech frequencies of 512, 1024 and 2048. The preoperative hearing level for speech is the average obtained from several, usually four, audiograms taken on different days.

usually four, preoperative audiograms), the best postoperative hearing result and the most recent hearing result, expressed in decibels gained or lost for speech as compared with the preoperative level. The hearing change in the ear not operated on is also recorded. This, to my mind, is important, since the intact ear may be regarded as the best control: A hearing gain as great in the intact ear as in the ear operated on cannot be ascribed to the surgical technic.

Study of the hearing change in the ears which were not operated on reveals an average variation of 4.6 decibels above or below the preoperative level, with an occasional extreme variation of 10 decibels, except for a case in which a gain of 11 decibels and another in which a loss of 27 decibels was shown. These observations are in agreement with the experience of others that there is a normal range of variation in repeated audiograms taken on a person with chronic deafness and that

the extremes of this range may, for practical purposes, be taken as ± 10 decibels. Therefore, in all analyses of results I regarded any change in hearing for the speech frequencies of less than 10 decibels as within the limits of normal variation. It will be observed that the control ears taken as a whole showed a slight tendency toward improvement, the

TABLE 3.—*Data on Twenty-Nine Ears Operated on by the Original Lempert Technic Modified by the Use of Constant Irrigation*

				Best Hearing Result			Final (Most Recent) Hearing Result			
Case No.	Ear	Date of Operation	Pre-operative Hearing Level for Speech, Decibels	Time After Operation, Mo.	Fistula Test	Decibel Change for Speech	Time After Operation, Mo.	Fistula Test	Decibel Change for Speech	Decibel Change for Speech in Intact (Control) Ear
14	R	2/ 7/40	57	2	++++	+30	22	++++	+29	+10
15	L	2/21/40	59	10	+++	+13	10	+++	+13	+ 2
16	R	2/28/40	88	1	++++	+28	5	++	+ 5	+ 1
17	R	3/13/40	57	5	++++	+34	20	++++	+32	+ 7
18	L	4/24/40	66	2	+++	+33	7	+++	+11	+ 1
19	L	5/24/40	60	1	++++	+35	18	+++	+25	+11
20	R	6/19/40	67	14	++++	+32	19	++++	+29	— 4
21	R	7/10/40	54	1	+++	+14	1	+++	+14	0
22	L	7/11/40	62	10	++++	+33	17	++++	+30	— 4
8	L	7/17/40	47	3	++++	+29	14	++++	+25	Operation
23	L	7/18/40	56	1	++++	+38	16	++++	+29	+ 1
24	L	7/24/40	47	5	++++	+27	20	++++	+12	— 4
25	R	8/ 1/40	49	1	++++	+19	8	+	+ 1	0
26	R	8/ 7/40	62	7	++++	+34	14	++++	+29	— 8
27	L	8/14/40	66	2	++	+18	8	++++	+ 9	+ 2
28	R	8/21/40	63	3	—	+34	15	++	+25	Operation
29	L	8/28/40	79	4	++++	+42	16	++++	+32	+ 5
30	L	9/ 4/40	57	8	++++	+40	18	++++	+32	0
31	R	9/ 5/40	74	2	+++	+42	13	++	+14	+ 4
32	L	9/30/40	63	2	++++	+21	7	++	0	— 2
33	L	10/ 3/40	56	1	+++	+34	13	++++	+16	+ 1
34	R	10/ 7/40	55	5	++++	+40	17	++++	+35	— 6
35	L	10/16/40	53	3	++++	+36	12	++++	+30	+ 3
36	R	10/17/40	61	4	++++	+34	12	++++	+31	— 1
37	R	10/21/40	46	7	++	+31	17	++	+15	— 3
38	R	10/23/40	42	4	++++	+24	14	++++	+20	— 8
39	R	10/24/40	48	6	+++	+21	12	+	+ 6	— 7
40	L	10/28/40	No hearing	12	++++	Hearing for 1024 frequency at 95 Db.	16	++++	0	—23
41	R	10/30/40	69	7	++++	+37	17	++++	+29	+ 9
42	R	11/ 6/40	63	2	+++	+28	6	+	+ 3	— 9

hearing gain averaging 1.6 decibels. This may be attributed perhaps to improvement with practice, perhaps to the fact that masking was not used and, with very great improvement in the ear operated on, hearing in the opposite ear may seem to improve because some tones are heard across by the ear which was operated on.

Of great importance in evaluating the results of the fenestration operation is the time of closure: Can closure of a fistula occur many

TABLE 4.—*Data on Sixty-Three Ears Operated on by Lempert Technic Modified by Use of Constant Irrigation and Dissecting Microscope*

Case No.	Ear	Date of Operation	Pre-operative Hearing Level for Speech, Decibels	Best Hearing Result			Final (Most Recent) Hearing Result			
				Time After Operation, Mo.	Fistula Test	Decibel Change for Speech	Time After Operation, Mo.	Fistula Test	Decibel Change for Speech	Decibel Change for Speech in Intact (Control) Ear
43	L	11/ 7/40	78	3	—	+23	13	—	+ 6	0
12	L	11/13/40	67	11	++++	+27	15	++++	+19	Operation
44	L	11/20/40	55	4	++++	+32	16	++++	+22	Operation
16	R	11/25/40	79	5	++++	+34	14	++++	+15	Operation
45	R	11/23/40	70	6	++++	+18	16	++++	+13	— 1
46	R	11/28/40	59	2	++++	+39	3	++++	+34	+ 5
47	L	12/11/40	58	2	++++	+33	12	++++	+31	+ 3
48	R	12/26/40	68	4	++++	+43	12	++++	+38	+ 8
49	L	12/30/40	54	3	+++	+22	14	++++	+ 7	+ 6
50	R	1/ 6/41	51	4	++++	+38	12	++++	+28	— 3
51	L	1/ 8/41	60	7	++++	+32	14	++++	+15	+ 1
52	R	1/ 9/41	56	4	++++	+33	10	++++	+29	+10
53	R	1/15/41	64	6	++++	+36	12	++++	+34	+ 1
54	L	1/27/41	72	4	++++	+35	7	++++	+30	+ 8
55	L	1/29/41	63	3	++++	+31	9	++++	+21	+ 6
56	L	1/30/41	55	3	++++	+28	13	++++	+18	— 5
57	L	2/ 6/41	93	11	++++	+30	12	++++	+26	— 8
58	L	2/12/41	44	4	++++	— 6	8	++++	—18	+ 1
59	R	2/17/41	50	6	++++	+35	13	++++	+27	+ 3
60	R	2/26/41	50	3	++++	+17	12	++++	+ 3	— 6
61	R	2/27/41	64	1	++++	+32	10	++++	+11	+ 7
62	R	3/12/41	46	4	++++	+18	6	++++	+13	+ 9
63	L	3/13/41	67	3	++++	+42	12	++++	+36	Operation
64	R	3/20/41	45	5	++++	+17	5	++++	+17	+ 5
65	L	3/24/41	50	1	++++	+23	12	++++	+15	Operation
66	R	3/31/41	54	1	++++	+34	11	++++	+31	+ 3
67	R	4/ 2/41	51	1	++++	+16	6	++++	+ 4	0
68	R	4/17/41	51	7	++++	+28	9	++++	+19	— 3
69	R	4/22/41	64	3	++++	+14	11	++++	+ 9	Operation
70	R	4/24/41	63	3	++++	+18	11	++++	+ 5	Operation
71	R	4/30/41	67	8	++++	+19	8	++++	+19	+ 1
72	L	5/ 5/41	51	2	++++	+34	6	++++	+28	+ 2
73	R	5/14/41	51	5	++++	+24	10	++++	+21	— 1
74	R	5/15/41	54	7	++++	+19	9	++++	+14	— 2
75	L	5/26/41	56	1	—	+18	6	—	— 4	+ 1
76	L	5/28/41	58	4	++++	+33	6	++++	+23	+ 1
77	R	6/ 9/41	67	8	++++	+35	8	++++	+35	+ 9
78	R	6/11/41	70	6	++++	+33	9	++++	+29	+ 3
79	R	6/12/41	49	4	++++	+22	6	++++	+21	+ 9
80	R	6/19/41	66	10	++++	+26	10	++++	+26	— 2
81	R	7/ 7/41	48	5	++++	+11	7	++++	+10	+ 1
82	L	7/ 9/41	57	3	++++	+29	6	++++	+20	+ 1
83	R	7/10/41	62	4	++++	+40	8	++++	+35	0
84	L	7/14/41	64	4	++++	+34	7	++++	+27	+ 6
28	L	7/16/41	64	4	++++	+37	4	++++	+37	Operation
85	L	7/17/41	54	1	++++	+24	1	++++	+24	+ 7
86	R	7/21/41	62	4	++++	+25	6	++++	+22	+ 1
87	R	7/24/41	37	3	++++	+17	8	++++	+ 6	— 9
88	L	7/28/41	58	2	++++	+30	6	++++	+18	+ 1
89	L	7/30/41	53	2	++	+21	7	—	+10	+ 6
90	R	8/ 2/41	59	4	++++	+27	5	+++	+19	— 2
91	L	8/ 4/41	52	4	++++	+20	7	++++	+ 7	— 7
92	L	8/ 6/41	69	2	++++	+41	6	—	+ 9	+ 4
93	R	8/ 7/41	35	4	++++	+17	7	++++	+10	— 2
94	L	8/18/41	42	3	++++	+18	7	+++	+ 8	+ 2
95	L	8/20/41	72	4	++++	+45	4	++++	+45	+ 9
96	R	8/25/41	54	2	++++	+34	6	++++	+26	+ 9
97	L	8/27/41	53	3	++++	+21	8	++++	+ 8	+ 1
98	L	8/28/41	54	3	++++	+31	3	++++	+31	+ 8
99	R	8/30/41	59	2	++++	+31	4	++++	+26	+ 2
100	R	9/ 3/41	54	1	++++	+36	6	++++	+29	+ 7
101	L	9/ 4/41	64	3	++++	+44	6	++++	+37	+ 4
102	R	9/ 8/41	60	2	++++	+42	6	++++	+37	— 1
103	L	9/10/41	62	2	++++	+27	7	++++	+19	— 1

months or years after fenestration, or, if closure is going to take place, will it always do so within a few months of the operation?

In tables 5 and 6 are analyzed the data on the time of beginning of closure, as evidenced by a decrease in the hearing in 26 ears in which

TABLE 5.—*Analysis of Cases in Which Complete Closure Occurred* *

Original Lempert technic	8 closures in 15 ears (53%)
Irrigation added to technic	6 closures in 29 ears (21%)
Microscopic inspection added to technic	12 closures in 63 ears (19%)

Time of Beginning of Closure As Evidenced by Beginning of Drop in Hearing		Time of Complete Closure As Evidenced by Return of Hearing to Preoperative Level	
Preoperative Month	Number of Cases	Preoperative Month	Number of Cases
1st	0	1st	0
2d	10	2d	4
3d	6	3d	5
4th	3	4th	4
5th	4	5th	3
6th	0	6th	2
7th	2 †	7th	3
8th	0	8th	2
9th	0	9th	0
10th	0	10th	1
11th	0	11th	1

* Drop in hearing to within 10 db. of preoperative level.

† In 1 of these cases beginning of closure was noted during the seventh postoperative month, but since no audiogram had been taken since the fourth month closure may have begun earlier.

TABLE 6.—*Analysis of Cases in Which Partial Closure Occurred* *

Original Lempert technic	1 in 15 ears (7%)
Irrigation added to technic	5 in 29 ears (17%)
Microscopic inspection added to technic	5 in 63 ears (8%)

Time of Partial Closure	
Postoperative Month	Number of Cases
1st	0
2d	2
3d	1
4th	2
5th	0
6th	1
7th	1
8th	1
9th	0
10th	0
11th	1
13th	1
15th	1

* Initial hearing gain followed by loss of more than 10 db., but leaving a significant improvement (10 db. or more above the preoperative level).

the fistula closed completely (loss of all the hearing gain) and in 11 ears in which it closed partially (loss of 10 decibels or more of the hearing gain). These tables indicate that closure of an operative fistula with the technic used is most likely to begin within the first three months after operation; it occasionally begins during the fourth or fifth month and only rarely begins as late as the seventh month. Closure became com-

plete in the majority of cases within six months of the operation, in some cases not until the seventh or eighth month and in only 1 case as late as the eleventh month. The conclusion seems justified that the osteogenesis and fibrosis which individually or together cause closure of a fistula, with loss of the hearing improvement, are most active during

TABLE 7.—*Analysis of Data on Ears Which Maintained Their Maximum Hearing Improvement* *

Original Lempert technic	3 in 15 ears (20%)
Irrigation added to technic	18 in 29 ears (62%)
Microscopic inspection added to technic	46 in 63 ears (73%)

* Improvement to within 10 db. of their highest level.

TABLE 8.—*Analysis of "Final" Hearing Results in Fifteen Ears Operated on by Original Lempert Technic*

Hearing improved (more than 10 db. for speech)	4	(27%)
Hearing unchanged (within 10 db. of preoperative level)	8	(53%)
Hearing worse (more than 10 db. loss)	3	(20%)

TABLE 9.—*Analysis of "Final" Hearing Results in Twenty-Eight Ears Operated on by Lempert Technic Plus Irrigation*

Hearing improved			21 (75%)
Gain for speech 10 to 19 db.	6	(21%)	
Gain for speech 20 to 29 db.	8	(29%)	
Gain for speech 30 to 35 db.	7	(25%)	
Hearing unchanged			7 (25%)
Hearing worse			0

TABLE 10.—*Analysis of "Final" Hearing Results in Fifty-Five Ears Operated on by Lempert Technic Plus Irrigation and Microscopic Inspection*

Hearing improved			42 (76%)
Gain for speech 10 to 19 db.	15	(27%)	
Gain for speech 20 to 29 db.	17	(31%)	
Gain for speech 30 to 38 db.	10	(18%)	
Hearing unchanged			12 (22%)
Hearing worse			1 (2%)

the first three postoperative months, become progressively less active during the next two months and finally tend to reach a state of equilibrium beginning about the sixth or the seventh postoperative month, but occasionally extending into the eighth to the eleventh month. A period of six months is the minimum that should elapse before a hearing result after fenestration is regarded as probably permanent.

In the analysis of "final" hearing results in tables 8, 9 and 10, only ears for which audiograms were taken more than six months after operation are included. Analysis of hearing results for ears for which audio-

grams were taken more than one year after operation (table 11) reveals no diminution in the proportion of good results as compared with those at the end of six months. This suggests that the result six months after fenestration may be regarded as the probable "final," or end, result, although there will be occasional rare cases in which the hearing will decrease as late as a year, or even fifteen months, after operation (table 6; fig. 11). It is of course impossible as yet to know with certainty the eventual outcome of the hearing in these cases five, ten or twenty years after fenestration.

The important question whether or not the production of a patent fistula will arrest the progressive nerve degeneration of otosclerosis cannot as yet be answered. While direct invasion of the cochlea or acoustic nerve accounts for this degeneration in a few instances, in the majority of cases its cause is not known. I have repeatedly observed a definite and considerable improvement in the bone conduction curve for the ear on which operation was performed, but this gain seems more

TABLE 11.—*Analysis of Hearing Results in Thirty-Six Ears One Year or More After Fenestration, with Use of One or Both Modifications*

Hearing improved			31	(86%)
Gain for speech 10 to 19 db.	8	(22%)		
Gain for speech 20 to 29 db.	12	(33%)		
Gain for speech 30 to 38 db.	11	(31%)		
Hearing unchanged			5	(14%)
Hearing worse			0	

likely to be due to increased mobility of the perilymph than to any regeneration of the nerve elements. It seems logical not to expect a fenestration to influence the progressive pathologic process of otosclerosis.

Tinnitus has not been relieved as invariably or as permanently in my experience as some authors have described. For instance, in case 29 the patient complained of terrific tinnitus in her left ear before operation, which was immediately and completely relieved by fenestration. Two months later, however, the tinnitus recurred and was as loud as ever, although the hearing improvement was unusually great and has been maintained for twenty months, with a strongly active response in the fistula test (fig. 12). One does not know what causes tinnitus, why fenestration should relieve it in most cases or why it should occasionally return.

Revision was carried out on 7 ears which lost their hearing gain after constant irrigation was added to the technic and on 1 ear after use of the microscope was introduced into the operation. As was observed

in the first cases of revision, failure of the fenestration was found to be due to closure of the fistula in every instance, but with this important difference: In the early cases the endosteal membrane was not completely removed and apparently acted as a protection to the contents of the horizontal canal, for in every revision the membranous labyrinth was seen to be floating freely in an open perilymph space, free from adhesions or fibrosis (table 1); in the later cases of revision, however, the perilymph space was invaded and often obliterated by bone or dense fibrous tissue, which was firmly adherent to the membranous labyrinth and

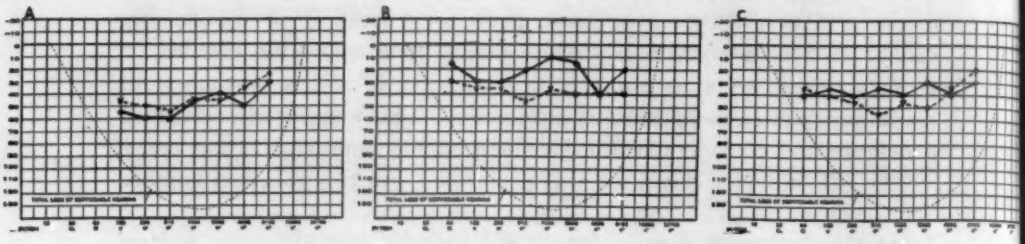

Fig. 11 (case 37).—Audiograms showing partial loss of the hearing improvement fifteen months after fenestration, probably due to bony narrowing of the fistula. This is the longest time after operation that a hearing loss has been observed.

A was taken before operation; *B*, seven months, and *C*, seventeen months, after fenestration operation on the right ear.

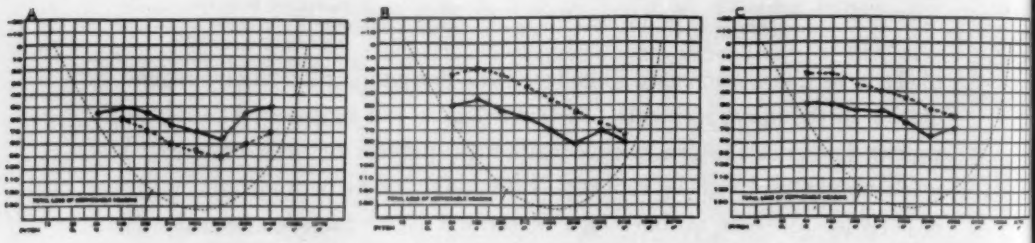

Fig. 12.—Return of severe tinnitus two months after fenestration, although a pronounced hearing gain of + 32 decibels for speech has been maintained.

A was taken before operation; *B*, two months, and *C*, sixteen months, after fenestration on the left ear.

sometimes compressed it to a thin cord for part of its course (table 12). To accomplish a successful revision in such cases the fistula must be extended forward to the ampulla, after removing the incus (novovalis technic), or a second fistula must be made into the superior semicircular canal. It is of considerable academic interest that a fistula into the superior (anterior) vertical semicircular canal always gives a pure vertical nystagmus on stimulation. A rotary nystagmus results only if

TABLE 12.—*Summary of Data on Revisions After Modified Fenestration Technic*

Case No.	Ear	Hearing Result at Time of Revision: Time After Operation, Mo.	Hearing Result at Time of Revision: Fistula Test	Hearing Result at Time of Revision: Decibel Change for Speech	Observations at Revision	Technic *	Final (Most Recent) Hearing Result: Time After Operation, Mo.	Final (Most Recent) Hearing Result: Fistula Test	Final (Most Recent) Hearing Result: Decibel Change for Speech
15	R	10	+	+13	Bone regeneration on surface of canal narrowing fistula; lumen of canal filled with dense fibrous tissue adherent to membranous labyrinth	Irrigation and micro.; 2d fistula made in superior canal	15	—	+19
16	L	5	++	+5	Bone regeneration on surface of canal obliterating fistula except for a small opening anteriorly; lumen of canal filled with new bone at one point, with dense fibrous tissue adherent to membranous labyrinth; membranous labyrinth accidentally avulsed	Thiersch graft; irrigation and micro.	14	—	—1
16	L	14	—	—1	Surface of canal unchanged; fistula wide open but completely filled with dense fibrous tissue; no sign of Thiersch graft except for three tiny encysted cholesteatomas on under surface of tympanomeatal flap	Irrigation and micro.; incus removed and novovalis type of fistula made	6	++	+18
18	L	7	+	+11	Fistula narrowed to slit by white bone, distinct from labyrinthine capsule; lumen of canal almost filled by new bone compressing membranous labyrinth	Thiersch graft; irrigation and micro.	16	—	—19
25	R	9	+	+1	Fistula closed by white bone, distinct from labyrinthine capsule except for tiny opening anteriorly; membranous labyrinth firmly adherent to bony lid posteriorly	Irrigation and micro.; 2d fistula made in superior canal	8	++++	+11
27	L	8	++++	+9	Fistula closed by white bone, distinct from labyrinthine capsule; lumen filled by this bone	Irrigation and micro.; 2d and 3d fistulas in posterior and superior canals	12	++++	+6
32	L	7	++	0	Fistula closed by white bone, distinct from labyrinthine capsule and adherent to membranous labyrinth	Irrigation and micro.; 2d fistula made in superior canal	8	++++	+33
42	R	6	+	+3	Operative notes lost.....	Irrigation and micro.	10	—	+8
92	L	6	—	+9	New bone on surface of bony canal almost obliterating the site of the fistula; dense fibrous tissue in lumen adherent to membranous labyrinth	Irrigation and micro.; incus removed and novovalis type of fistula made	2	—	+36

* "Micro." indicates that use of the dissecting microscope was introduced into the operation.

there is a positive response to the fistula test for the horizontal canal also and if both canals are pressed on simultaneously.

The results of revision are analyzed in tables 13, 14 and 15. It will be observed that a revision done within eight months of the fenestration is more likely to result in closure than a revision done later. This accords with the idea that osteogenesis and fibrosis are active during the early postoperative months and a revision done during this period

TABLE 13.—*Summary of Results of Revision* *

Hearing improved	17
Hearing unchanged	2
Hearing worse	1

* One revision, 20 ears; two revisions, 2 ears.

TABLE 14.—*Relation of Time After Operation to Final Results of Revision*

Time of Revision After Operation	No. of Ears	No. of Ears With Improved Hearing	Percentage of Ears with Improved Hearing
4 mo.	1	0	0
5 mo.	1	0	0
6 mo.	2	1	50
7 mo.	2	1	50
8 mo.	1	0	0
9 mo.	2	2	100
10 mo.	2	2	100
1 yr. or longer	9	9	100

TABLE 15.—*Relation of Technic of Revision to Final Results of Revision*

	With Thiersch Graft	Without Thiersch Graft	Original Fistula Reopened	New Fistula Made: Superior or Posterior Canal	New Fistula Made: Into Bony Ampulla (Novovalis Technic)
Hearing improved	5	9	7	1	3
Hearing unchanged	2	2	3	1	0
Hearing worse	1	0	1	0	0

will serve only further to stimulate this activity, whereas after eight months the tissues have reached a state of equilibrium comparable to that existing before the first operation. The use of a Thiersch graft did not improve the results of revision. When a new fistula was made into another semicircular canal or into the bony ampulla (novovalis technic) the results were better than when the original fistula was merely reopened.

In the last analysis the final hearing result for each patient is what counts, and these results are summarized in tables 16 and 17. The practi-

cal value to the patient of a hearing improvement is not easily measured. Experience has shown that when hearing for the speech frequencies is restored to within 20 decibels of normal the patient will usually regard himself and will be regarded by his friends as having essentially "normal" hearing. When the hearing for speech is restored to within 30 decibels of normal the patient will be able to meet the majority of social and economic situations without a serious handicap. Thus, a 12 or 14 decibel

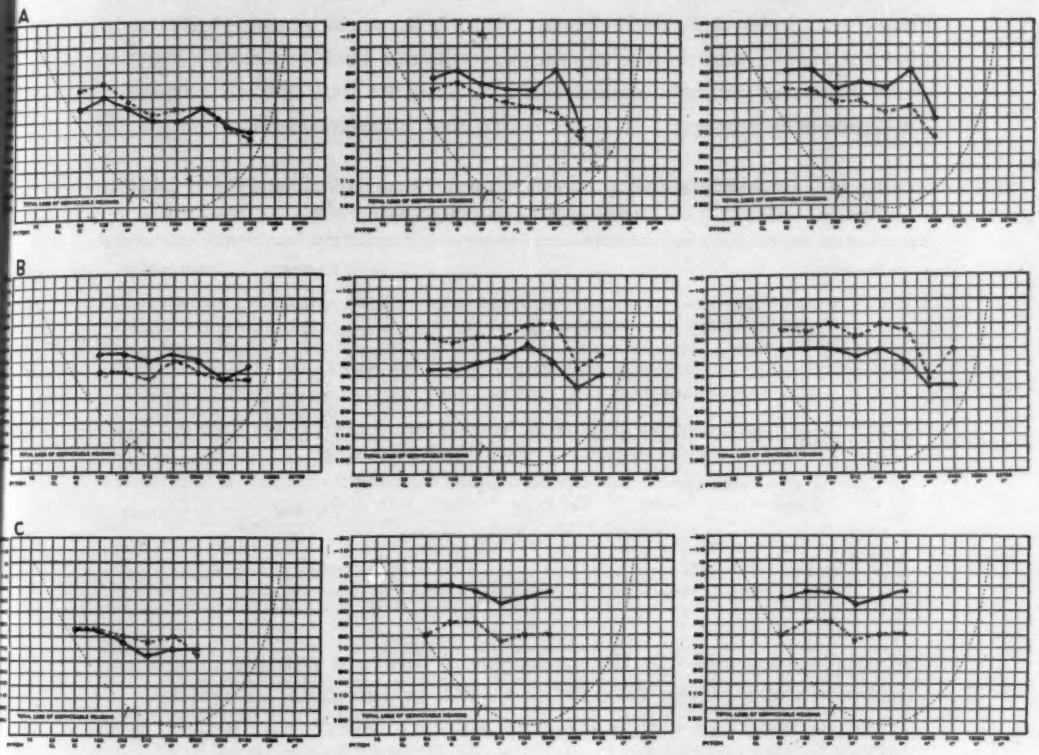

Fig. 13.—*A* (case 17), hearing gain of + 29 decibels for the speech frequencies maintained for twenty-five months after modified fenestration technic. The audiogram on the left was taken before operation; that in the center ten months after, and that on the right twenty-five months after, fenestration on the right ear.

B (case 30), hearing gain of + 32 decibels for speech maintained for eighteen months after modified fenestration technic. The audiogram on the left was taken before operation; that in the center one year after, and that on the right eighteen months after, fenestration on the left ear.

C (case 48), hearing gain of + 38 decibels for speech maintained for sixteen months after modified fenestration technic. The audiogram on the left was taken before operation; that in the center one year after, and that on the right sixteen months after, fenestration on the right ear.

gain may be satisfactory for a patient with a 40 decibel loss for the speech frequencies, whereas a 30 decibel gain would be necessary to restore a patient with a 60 decibel loss to the category of a hearing person. However, I do not agree that patients with a profound loss of hearing of 70, 80 or 90 decibels should be refused operation on the grounds that their hearing cannot be restored to a 20 or 30 decibel level. It has been my experience that a 30 decibel gain, even though it does not give "practical" hearing, may mean as much to such profoundly deafened persons as to those with a 40 or 50 decibel loss. Among my most grateful and happy patients are 2 who before operation could not hear any word shouted into either ear but who can now understand the loud spoken voice at a distance of a few feet. It is necessary that bone conduction hearing be

TABLE 16.—*Hearing Status of One Hundred and Three Patients at Time of Writing (April 1942) as the Result of One Hundred and Forty-One Operations*

	Number	Percentage
Hearing improved	87	84
Hearing unchanged	13	13
Hearing worse	3	3

TABLE 17.—*"Final" Hearing Results in Ninety-One Patients Given Hearing Tests Six Months or More After Operation*

	Number	Percentage
Hearing improved	75	83
Hearing unchanged	13	14
Hearing worse	3	3

relatively good and that these profoundly deafened patients understand that the most they can hope for is a small improvement in hearing.

It has been said with more justification, I believe, that persons with slight losses of hearing should not be operated on. If the hearing for speech is within 30 decibels of normal in the better ear, it is doubtful whether fenestration will give enough practical improvement to be worth the risk, for there is always the possibility that the hearing might be made worse instead of better. For this reason, I adhere strictly to the rule never to operate on the better ear. I have also attempted to profit by Lempert's experience and teaching and do not operate on patients with primary nerve deafness or those with poor bone conduction. Such results as I am able to report are due in considerable part to careful selection of cases and to the avoidance of overenthusiasm for the operation. Every patient who proves to be a suitable candidate for the operation is told exactly the chances of success, the chances of failure and the chances for the hearing's being made worse in the ear operated on.

Complications, operative and postoperative, are summarized in table 18. I have been fortunate in having had no serious surgical complications, no fatalities, immediate or late, and no dead labyrinths in 194 consecutive operations on 154 patients from July 1938 to the date of writing (April 1942).

The data which I have presented are intended to be sufficiently complete to form a basis for comparison with results obtained with other technics. It is to be hoped that future reports of results may be complete enough to permit an accurate comparison of hearing results, for in no other way will one be able to judge the relative merits of the different technics, and thus to continue the development and improvement of the surgical treatment of otosclerosis.

TABLE 18.—*Complications and Untoward Results in One Hundred and Thirty-Seven Consecutive Operations*

	No. of Cases
Membranous labyrinth in the horizontal canal destroyed	1
Tympanomeatal flap torn; fragments of skin from anterior meatal wall used to cover fistula	2
Tear in dura with spinal fluid leak during operation	3
Facial paralysis beginning 1 week after operation, with complete recovery within 1 to 2 weeks	2
Dizziness, consisting of sense of unbalance on motion, lasting more than 1 year	3
Postoperative atelectasis	1
Postoperative psychosis coinciding with hearing improvement and attributed by neurologist to removal of a "crutch" on which the patient had leaned to escape unpleasant responsibilities	1
Dead labyrinths	0
Death or serious complications	0

SUMMARY AND CONCLUSIONS

With the original fenestration technic a lasting and significant hearing improvement was obtained in about 25 per cent of cases.

With a modified fenestration technic in which the use of constant irrigation seems to be the most important improvement, a lasting and significant hearing improvement was obtained in about 75 per cent of cases.

Six months is the minimum period that must elapse after operation before a hearing result is regarded as "final" and probably permanent. Complete closure of a fistula rarely begins later than six months after operation, but partial closure, with partial loss of the hearing improvement, may be observed as late as fifteen months after operation. The osteogenesis and fibrosis causing closure of a fistula are most active during the first three postoperative months and in most cases tend to reach a state of equilibrium about six months after operation.

In every revision failure of the operation was found to be due to closure of the fistula by new bone or fibrous tissue.

At least six, and preferably eight, months should be allowed to pass before one attempts a revision in order to permit the active osteogenesis and fibrosis to reach a state of equilibrium.

With the revisions and with operations on both ears in some instances, a significant hearing improvement was secured in 87 of 103 cases in which operation was performed between July 1938 and September 1941. The average hearing gain in these 87 cases was 24.4 decibels for the speech frequencies.

No dead labyrinths, no serious complications and no fatalities resulted in a series of 194 consecutive operations in 154 cases from July 15, 1938 to April 24, 1942.

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VASOMOTOR RHINITIS IN THE TROPICS

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Vasomotor rhinitis is a well known symptom complex, characterized by swelling of the turbinates, particularly the inferior ones, attacks of sneezing and watery rhinorrhea. Occasionally headache and conjunctivitis are present. In some cases an allergic origin has been demonstrated; in others such an origin is questionable. The prognosis is rarely serious, but a complete cure is difficult to attain. Occasionally the condition is seen in combination with asthma.

In the last two and a half years I have treated 130 patients with vasomotor rhinitis in the republic of Honduras, in Central America. Two of these also have asthma. A comparison has been made between the patients I treated in Berlin from 1931 to 1933 and those I have treated in the highlands and along the coast of Honduras. The conditions at this outpost of otolaryngology are not proper for a scientific or statistical study, and a review of the literature cannot be made at present, but certain of the following observations on vasomotor rhinitis in the tropics should be of value.

On the whole, only a minority of my patients have been of the European race; most of them were mestizos, a cross breed of Indians and Europeans, often, especially on the coast, mixed with Negro, sometimes with pure Negro, cross-bred with Chinese. The ages of these patients ranged between 6 and 79 years, with the majority between 20 and 40 years. The sexes were equally affected.

Tegucigalpa is located in a high mountain valley, 914 meters above sea level. The climate is cool for a tropical region, and the vegetation is richer in pine trees than in palms and banana trees. From March until May it is hot, and during this period there is usually no rain. The streets are in poor condition and are very dusty. The general hygiene is poor. When I started to work in this region, I found a higher percentage of patients with the symptom complex of nasal occlusion, sneezing and watery rhinorrhea than I had observed in Berlin. Rhinoscopic examination usually showed the turbinates to be swollen, bluish and pale or red. Occasionally no abnormality could be seen. The symptoms were normally more pronounced in the morning, during the rainy season or in association with a change of temperature.

When I came to San Pedro Sula, near the Atlantic coast of Honduras, there seemed to be more patients with vasomotor rhinitis. The climate

and vegetation are quite different from those of Tegucigalpa. San Pedro Sula is 60 meters above sea level, and the temperature is typical of the tropics, with much rain, great heat and dampness. There is less dust than in Tegucigalpa. In both places the patients' symptoms and the clinical findings were the same. Directly on the coast, in the two ports of Puerto Cortés and La Ceiba, vegetation is more or less the same as in San Pedro Sula. There is about as much dust as in Tegucigalpa, and general hygiene is also very poor. I had the impression that vasomotor rhinitis is not less frequent there and that the sea breeze does not change conditions.

There are no facilities for the study or the treatment of allergy. It is assumed that most of the cases of vasomotor rhinitis are of allergic origin. Many patients say that they feel better when they are outside the dusty city.

Some people from the United States have complained of nasal obstruction and sneezing during their visit in Tegucigalpa, although I recall that a patient from Long Island, N. Y., who suffered regularly during the ragweed pollen season while at home was free from trouble in Tegucigalpa except for slight dyspnea on exertion before the rainy season. There are some cases in which it is difficult to establish an etiologic relation to allergens and others in which the causative agent remains obscure. A few interesting cases illustrate this.

A 20 year old married woman had left San Pedro Sula for the southern part of the United States at 12 years of age. At 15 years of age her tonsils were removed. After this procedure, tendency to have frequent colds developed. At the age of 18 years she returned to San Pedro Sula, where a chain of familiar symptoms developed—severe attacks of sneezing in the morning, watery rhinorrhea and nasal occlusion. These attacks did not cease during sea voyages, and diagnostic washing of her sinuses as well as roentgen examination revealed no abnormalities. There were no signs of hysteria, neurasthenia or nervousness. She was completely relieved of her symptoms when she was treated, as will be described later in this paper.

In this case the observations did not fit the usual clinical picture of allergy, as the symptoms continued when the patient was on a sea voyage.

Urbach [1] found few patients with a family history of allergy. This was my experience, and in no case was vasomotor rhinitis seen in combination with urticaria or other cutaneous manifestations. There were only 2 patients with vasomotor rhinitis and asthma combined. One of these was a 30 year old woman who was relieved of both her asthma and her vasomotor rhinitis after treatment in the manner about to be described. The other patient was a 53 year old pharmacist who had suffered with asthma and vasomotor rhinitis for many years and who

1. Urbach, E.: Vasomotor Rhinitis, Arch. Otolaryng. **33**:982 (June) 1941.

had not been able to sleep for two weeks on account of asthma and nasal occlusion. His nasal septum had been operated on, and he had had polyps removed on several occasions. After the first day of the treatment recommended, he was much improved, and after one week of this therapy he felt well. Both patients lived on the coast and were treated in San Pedro Sula, so that a change in climate cannot be considered the cause of the recovery.

Benjamins [2] rarely found asthma connected with tropical vasomotor rhinitis. Further observations will be necessary to determine whether the combination of asthma and vasomotor rhinitis is as rare as it has been in my experience. Benjamins studied vasomotor rhinitis in the Dutch East Indies. He called the condition "rhinitis spastica vasomotoria tropica" and identified it with "tropical hay fever." He considered the disease in most cases due to pollen sensitivity. Among other etiologic factors, he mentioned the dust of *padi* (unpeeled rice), tea and copra and stated that "the other forms of nervous rhinitis" were to be found. He observed, as I have in Honduras, that the disease occurs along the coast and in the mountainous areas and that the change of climate in the country or moving to a temperate zone is an important factor in the cure. He did not mention in his writings whether most of his patients were Europeans or natives, but he stated that some Europeans lost their hay fever in the Indies, whereas some who had never experienced nasal symptoms contracted "tropical hay fever." My observations were mostly on natives or part-Europeans. However, as yet I cannot find a difference between the two races to account for the difference between them in disposition or in clinical aspect.

If the observation is true that vasomotor rhinitis is found more frequently in Honduras than in Berlin, it is obvious that there should be a reason. The allergens in question are most probably pollen of tropical vegetation and dust. In this series there were no cases in which occupational contact with allergens could be suspected. Neither did psychic disturbances seem to play an important role, as they did in some of Urbach's cases. Intestinal parasites are among the most common causes of disease, which many people are never rid of. Purgatives and other medicines are abused. Thus intestinal factors of allergic or toxic origin may be involved too, even if this did not become obvious in my cases. Intestinal troubles may contribute to weaken general resistance, as well as chronic malaria and poor nutrition, particularly that due to lack of vitamins. I do not know whether there is any exact knowledge about the influence of insufficient nutrition or of specific vitamins in

2. Benjamins, C. E.: Die Rhinitis spastica vasomotoria tropica, in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1928, vol. 4, p. 666.

vasomotor rhinitis, but the great majority of the people of Honduras are accustomed to anything but a well balanced diet. Their meals consist chiefly of beans, rice, *tortillas* (corn meal) and meat. The manner of preparing meals is primitive. Deeks,[3] an expert on health problems of tropical America, concluded that many of the difficulties encountered in the management of health problems in tropical countries rest on dietary deficiencies.

Many of the patients in this series had received various kinds of therapy, including the administration of nose drops and cold vaccines, electrocauterization of the turbinates, turbinectomy and submucous resection. Most of these treatments were useless. Correction of a septal deformity causing obstruction was of value in that it created an adequate airway, but it in no way alleviated the sneezing or the rhinorrhea. One patient had a Caldwell-Luc operation but after operation continued to have the same complaints. His condition improved on the therapy to be described. No vasomotor rhinitis has been seen which has been connected etiologically with sinusitis, otitis media or tonsillitis. In general, it has been observed that operations are of no value in the direct treatment of vasomotor rhinitis, and conditions which may be a contributing cause and warrant operation are seldom found. Urbach made the statement that operations for the treatment of vasomotor rhinitis "are usually without lasting effect; occasionally they are even harmful." If real hypertrophy of the turbinates is present and the turbinates do not shrink after application of a vasoconstrictor, the removal of the hypertrophic tissue may be indicated. I have not performed this operation either in Europe or in the tropics. Recently, Proetz [4] stressed the necessity of dealing with intranasal conditions on a functional rather than a structural basis. Piness and Miller [5] stated that symptoms of allergy are frequently confused with those of infection. In my experience, vasomotor rhinitis is not alleviated by tonsillectomy. The upper respiratory tract in children seems to become more irritable after the operation. Yearsley [6] regarded many tonsillar operations as "a slaughter of the innocents which out-Herods Herod," and this procedure is never less indicated than in vasomotor rhinitis.

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3. Deeks, W. E.: Health Problems in Tropical America with Particular Reference to Diet Deficiencies, in Arbeiten über Tropenkrankheiten und deren Grenzgebiete, Hamburg, Kommissionsverlag L. Friederichsen & Co., 1927, p. 62.

4. Proetz, A. W.: The Functional Point of View in Rhinology, J. A. M. A. **115**:421 (Aug. 10) 1940; abstracted, Arch. Otolaryng. **33**:323 (Feb.) 1941.

5. Piness, G., and Miller, H.: Allergy of the Upper Respiratory Tract in Infancy and Childhood, J. A. M. A. **113**:734 (Aug. 26) 1939; abstracted, Arch. Otolaryng. **33**:480 (March) 1941.

6. Yearsley, M.: The Present Position of Tonsil Operations, Brit. J. Child. Dis. **36**:1 (Jan.-March) 1939; abstracted, Arch. Otolaryng. **33**:478 (March) 1941.

The following treatment is one which I have used and found satisfactory both in Europe and in the tropics. I do not imply that it is either new or exclusive. The procedure is as follows: Anesthetize the inferior turbinates with either 2 per cent pontocaine hydrochloride–epinephrine or 10 per cent cocaine–epinephrine solution, and then superficially cauterize them with a concentrated solution of trichloroacetic acid. Calcium gluconate is given by intramuscular injection; in cases in which the discomfort is acute, it should be given by the intravenous route. Ephetonine (phenylmethylaminopropanol hydrochloride) is given by mouth. Many similar preparations may work as well as ephetonine, but my experience with them is too limited for me to evaluate them properly. This type of therapy seldom causes trouble.

A very few patients complained of "feeling bad" or had fever after the treatment, and it was difficult to judge whether there was a concomitant infection or recurrence of a tropical disease or whether the condition was due to the treatment itself. The great majority of patients had no unfavorable reactions and were soon free of the vasomotor rhinitis. Some patients experienced a relapse five to sixteen months after the treatment and were again relieved of their symptoms by the same therapy. Two patients experienced relief from sneezing and rhinorrhea, but nasal occlusion persisted on the side of a septal deflection. These symptoms were alleviated by submucous resection.

In general, septal deflections in patients with vasomotor rhinitis should not be treated surgically before the conservative therapy recommended has been tried.

For a patient whose septal deflection is so near the turbinates that there is danger of adhesions after the use of a caustic, a septal operation is indicated. All these operations should be preceded by treatment with calcium and ephetonine, to reduce the irritability of the nasal mucous membranes. Benjamins [7] recommended that treatment with calcium be given before all operations in the tropics because he found a greater disposition for hemorrhage there than in temperate zones. Up to the present I cannot confirm this for Honduras.

One of the many medical prejudices of the people of Honduras is the fear that any disease will be made worse by baths. Baths in this country are for the most part cold showers, and my patients were advised to continue taking these along with their treatment. In a tropical climate, physical hygiene with thorough cleansing of the pores is more important than elsewhere, and the regular use of cold showers contributes to an improvement in the general body tone. These patients were also advised

7. Benjamins, C. E.: Die Tropenkrankheiten der Luftwege und der Mundhöhle, in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1928, vol. 4, p. 612.

to use a diet rich in vitamins, which can be provided easily in this country at a very low cost.

Treatment with specific pollen extracts cannot be carried out here at the present time. If the problem which prevents this can be solved, Benjamins'[2] warning should not be forgotten—that specific pollen extracts should be administered with the greatest caution, because people in the tropics are living in an environment rich in pollen. Pollen extracts from other countries are of no value.

SUMMARY

The observations and impressions in about 130 cases of vasomotor rhinitis in the republic of Honduras, in Central America, are reported. These observations were made in the mountainous areas as well as in the coastal districts of this tropical country. A warning is given against unnecessary operations, and a method of conservative therapy is proposed, which has been found to be satisfactory.

CLINICAL ASPECTS OF FUNCTIONAL HOARSENESS

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Much has been written about pathologic conditions of the larynx and the accessory organs of phonation, and especially about acute and chronic hoarseness, for which many possible etiologic factors are given. It is unnecessary to enumerate here these various disturbances, since the present paper is concerned only with "functional hoarseness."

Pryor [1] remarked concerning phonatory disturbances:

> To the general practitioner the larynx has always been more or less of a land of mystery. Up until a few years ago there was probably less known about this organ by ear, nose and throat specialists than any other one single phase of their work.

In 1941 these remarks still hold true in regard to the condition of functional hoarseness. In glancing through the literature on the subject one finds many promising attempts to uncover the mystery surrounding functional hoarseness, but nowhere a full explanation and statement of its treatment.

Lewin, in 1862, as reported by Mes,[2] mentioned the condition called "clergyman's sore throat." Lewin's findings, including the results of laryngoscopy, led him to the conclusion that this condition is due to a faulty phonatory mechanism. The symptoms were given as weakness of the voice, easy fatigue, dry throat, slight pain in the throat and prickling sensations. Imhofer, who was mentioned in the same paper,[2] called the condition a disturbance of coordination which hinders the law of "a minimum of effort with a maximum of result." Mes recommended as treatment breathing exercises and speech lessons, but no further details as to how they should be carried out were given.

Brueggemann [3] expressed the opinion that phonatory laryngeal disturbances without organic defects of the larynx are especially important for the clinician. He likewise recognized faulty speech and phonation as a cause but did not give any details or advice as to treatment.

From the Woman's Medical College of Pennsylvania.

1. Pryor, W. R.: A Practical Consideration of Alterations of the Voice, Kentucky M. J. **32**:415-418 (Aug.) 1934.

2. Mes, L.: Functional Hoarseness, Nederl. tijdschr. v. geneesk. **75**:3990 (July 25) 1931.

3. Brueggemann, A.: Die Heiserkeit und ihre Behandlung, Med. Klin. **29**: 1099-1100 (Aug. 11) 1933.

Tilley [4] spoke of functional aphonia as a common type of loss of phonation and said it was due to paresis of the adductor muscles of the vocal cords. He suggested as treatment some powerful suggestion, or even unexpected shock, application of Mandl's solution (pigment) to the glottis, passage of a strong faradic current between the intralaryngeal electrode and the external terminal, administration of a tonic containing strychnine or physical shock.

Nadoleczny [5] agreed that functional hoarseness is due to faulty technic in phonation or strain on the phonatory apparatus. He advised speech lessons, training of the phonatory mechanism and psychotherapy.

Negus,[6] of London, stated:

> One affection of hoarseness affecting women is functional aphonia. The reason for the upset is inability to bring the vocal cords together on attempted phonation. Air from the lungs escapes through the partially or completely open glottis.

No treatment of the condition was mentioned.

Liggett [7] described the action of the extrinsic and intrinsic muscles of the larynx. The extrinsic muscles move the larynx as a whole in fixing the cartilages to improve the vibratory action of the intrinsic muscles. The intrinsic muscles move the vocal cords. All intrinsic muscles are adductors of the vocal cords with the exception of the posterior cricoarytenoids, which are abductors. A functional type of hoarseness due to a psychogenic disturbance must be differentiated from hoarseness due to an organic lesion.

Wells [8] published a paper on hoarseness.

> It is sometimes necessary to warn our public speaking patients that mere loudness of the voice is not all that is needed to make themselves heard and understood. Clearness and intelligibility of speech depends to a great extent upon good articulation and accurate production and vowel resonance.
>
> With proper vocal action one can speak with little effort.
>
> . . . A good voice depends on (1) good formation and a healthy condition of the cords; (2) good development of the muscles activating the cords; (3) adequate lung power; (4) favorable development of the resonance cavities of nose, mouth and throat, and (5) proper methods of articulation. Continuous improper

4. Tilley, H.: Chronic Hoarseness: Its Clinical Significance, Diagnosis and Treatment, M. J. Australia **2**:169-174 (Aug. 5) 1933.

5. Nadoleczny, W.: Wodurch kann eine chronische Heiserkeit bedingt sein? München. med. Wchnschr. **82**:13-16 (Jan. 3) 1935.

6. Negus, V. E.: The Significance of Hoarseness, New York State J. Med. **39**:9-12 (Jan. 1) 1939.

7. Liggett, H.: The Mechanism of Hoarseness: Its Medical and Neurological Aspects, M. Clin. North America **22**:1463-1472 (Sept.) 1938.

8. Wells, W.: The Significance of Hoarseness, Ann. Otol., Rhin. & Laryng. **49**:99-112 (March) 1940.

use . . . should be suspected in those who complain of voice fatigue following singing exercises, or who speak in an unnatural cramped method, or who have poor resonance, and articulate indistinctly.

He recommended training from a speech teacher.

Froeschels [9] listed the following characteristics of faulty phonation:

1. Weak, hoarse voice, general lack of energy and absence of tonus of the whole phonatory apparatus, with uncontrolled passage of air.
2. Muffled voice, with the jaws unopened and the lips and tongue immovable.
3. Suppressed sounds, especially the vowel "a," with the root of the tongue raised and pressed on the entrance of the glottis.
4. Flat, almost harsh voice, as though formed in the region of the palate in wide mouth opening, the soft palate being contracted and the tongue drawn backward.
5. Throaty sounds, similar to those of a trumpet, and compression of the pharynx and the laryngeal entrance.
6. Hoarse, compressed sounds and increased compression of the entrance of the glottis, especially of the ventricular bands.
7. Various types of nasal voice.
8. Bleating voice, consisting of a combination of exaggerated nasal and throaty tones.

Schroeder [10] remarked that in cases of functional hoarseness the physician feels immediately that the cause is an entirely faulty phonation. Have the patient read a few lines with loud voice or sing a few tones and one hears the totally faulty tone formation. The whole load is put on the vocal cords and the larynx; the consonants are pronounced indistinctly; no use is made of the anterior part of the *Ansatzrohr* (lips, tongue and facial musculature). Cure is possible only by complete reeducation in use of the speech and singing mechanism; correct phonation is the only helpful therapy. But how can this be accomplished? There is great controversy about the formation of the tone. Some advise the whistle position of the *Ansatzrohr,* like a megaphone. Indrawing of the cheeks and dropping of the lower jaw is the most important point in the method, then development of the voice from the piano, the "head voice" out, and further development of the middle and the chest voice.

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9. Froeschels, E.: Gesetzmässigkeiten in der Erscheinung und Entwicklung der hyperfunktionellen Heiserkeiten, Monatschr. f. Ohrenh. **7**:400-405 (April) 1937.

10. Schroeder, T.: Ueber Heiserkeit infolge verkerhrter Stimmbildung und deren Heilung, Monatschr. f. Ohrenh. **66**:343-345 (March) 1932.

Voelker [11] advised that the patient be trained to extend the superior laryngeal aperture and relax the sphincter for articulation. The best means of doing this is to open the mouth and take a full inspiration, as in yawning. Then, while maintaining a relaxed laryngeal sphincter, the patient should practice respiratory gymnastics, then whispering the vowel sounds and, finally, vowel phonation, with gradual building up of consonants, syllables and words.

Felderman [12] said:

> First and foremost we have the powerful resonators, known as the human lungs. The stronger the breathing apparatus the more powerful is the voice. Then we have the vocal apparatus. This consists of the vocal cords and their adjacent structures. When the expiratory blast of air is released from the lungs, that amount of air is gradually shaped by playing upon the vocal cords and exciting them into action or vibration, which in turn is reinforced by the resonating chambers, such as the nose and throat and the bony cavities of the face and the cranium. These vibrations are received by the articulators. Here the lips, tongue and teeth play a conspicuous part in producing speech. One must not overlook the brain. Some authors believe that a human being can be compared to the telegraph system. When the above five factors are intact, no interference is possible unless some pathologic condition exists. When the physiologic action is faultless, the human speech is normal. On the other hand, should any of these five principles break down or not function correctly, then the speech becomes defective, the degree depending largely on where the defect exists, and to what extent.

In a review of a few papers recently published by nationally famous laryngologists, statements were found such as the following excerpts from a paper by Jackson [13]:

> Myasthenia laryngis is a name . . . applied to a morbid entity characterized by asthenia of the phonatory musculature of the larynx, especially the powerful overworked thyroarytenoideus muscles. It differs clinically from the ordinary fatigue of the muscle . . . in that exercise does not strengthen the weakened muscle.
>
> Damage to the thyroarytenoideus muscles is nowhere recognized as the fundamental pathologic factor in the ruin of the great singer's voice.
>
> There is direct trauma in the phonatory contact of the cords and in the friction of the passage of the air current up between them.
>
> All the anterior muscles of the neck are involved in the function of fixation and taking up of the slack.
>
> The walls during phonation become rigidly fixed; the higher the pitch, the greater the rigidity.

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11. Voelker, C. H.: Phoniatry in Dysphonia Ventricularis, Ann. Otol., Rhin. & Laryng. **44**:471-473 (June) 1935.

12. Felderman, L.: The Human Voice: Its Care and Development, New York, Henry Holt & Company, Inc., 1931.

13. Jackson, C.: Myasthenia Laryngis, Arch. Otolaryng. **32**:434-463 (Sept.) 1940.

Treatment.—Cure is obtainable only in the early stage of the disease. . . . No training or special exercise can be carried out without working the already jaded muscles. . . .

The best advice to give the great singer is to retire before disastrous failure causes lifelong regret.

Prognosis.—. . . The prognosis as to the future career is hopeless.

Conclusions.—It is a muscular disability as distinguished from a mucosal inflammation. The condition is really a muscular disability involving most severely the thyroarytenoid muscles.

One must conclude from these papers that the physician does not consider functional hoarseness as his field. Many of the authors recognized the faulty part; some outlined the treatment, but apparently they did not consider correction of phonation as part of their work. Most physicians will not send a patient to a physical therapeutist without a definite outline and prescription of what has to be done, the length of treatment, the dosage, etc. These physicians are at least familiar with all procedures and their applications. Speech correction may be a field of its own, and not always in the line of the ear, nose and throat specialist, but the physician should be familiar at least with the mechanism of the normal and the abnormal phonatory apparatus in order to make the right diagnosis and to supervise the speech teachers to whom he sends his patients. If the medical profession would adopt this attitude many young voices could be saved not only for a career of singing but from the standpoint of general health, and many of the "speech teachers" would disappear because of malpractice.

ANATOMY AND PHYSIOLOGY OF FUNCTIONAL HOARSENESS

Definition.—Functional hoarseness is a symptom complex of the laryngeal apparatus with involvement of the surrounding structures, attended by hoarseness caused by an incorrect phonatory mechanism as the most outstanding symptom. Hysterical aphonia should be excluded.

Functional hoarseness, like any condition concerned with phonation, includes not only the larynx but the whole respiratory system, the oral cavity, the accessory sinuses and the bones of the skull. Therefore, one must include in the discussion the anatomy and physiology of these structures in order to understand the proper function of phonation.

Respiration.—"If a moving column of air be partially or completely interrupted in its exit from an aperture in a rhythmical manner and at a rate within the limits of human audibility, musical sounds will be produced" (Negus [14]). This is the start of phonation. As one knows,

14. Negus, V. E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929.

the diaphragm, assisted by the intercostal muscles, is the motor in respiration. Watch a baby and one will see that the respiratory wave is mainly initiated in the diaphragmatic region, assisted by the abdominal muscles. In taking pneumographic tracings of the thoracic and diaphragmatic waves (figs. 1, 2 and 3), one finds that the two are almost equal; the diaphragmatic wave may even be greater. The same observation can be made on animals. This natural breathing sets the air

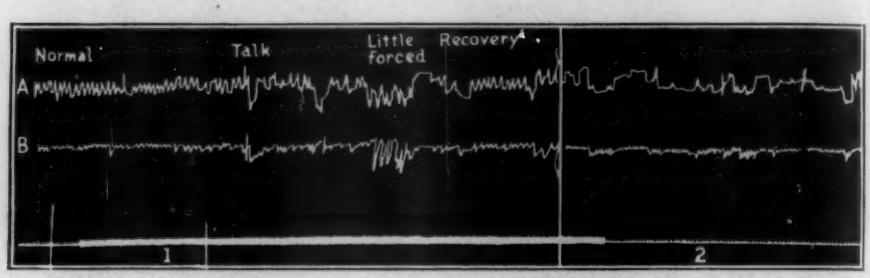

Fig. 1.—*1*, pneumographic tracings, (*A*) thoracic and (*B*) diaphragmatic, of a boy 5 years old. *2*, pneumographic tracing of a boy 2 years old.

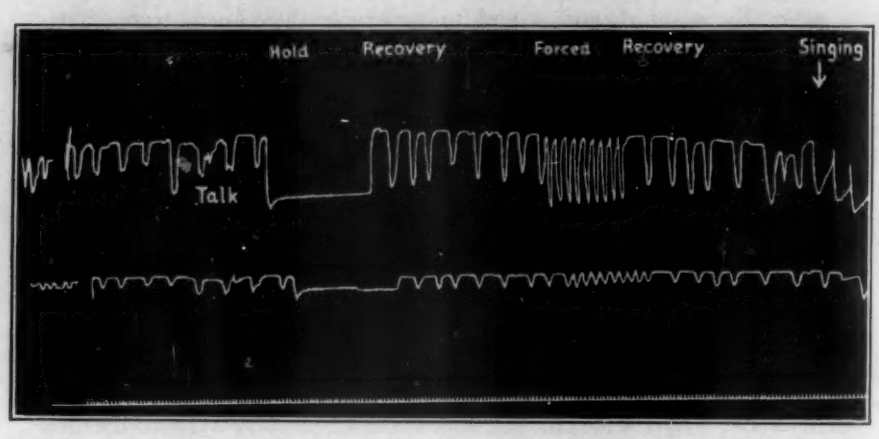

Fig. 2.—Pneumographic tracing of a woman aged 40 with a trained voice.

column into vibration and controls the amplitude and strength of the air column. When a baby cries, it may cry for hours; a cat may meow for hours; a dog may bark for a long time without getting hoarse, and this is taken for granted. The baby stops crying after the throat gets dry from the excessive intake of air combined with the open mouth breathing. One wonders why the child does not become hoarse like an adult, who would start to clear his throat in a few minutes if he should try to imitate the child's cry. But here is one of Nature's secrets: the

baby uses all its organs in an absolutely natural way; breathing and crying are brought about like sucking, defecation and sleep, as simple reflexes of the subcortical centers. The baby does not think why and how it is breathing. This natural function will not be changed until the cortex is fully developed. This development and the ability to "think" are of advantage to the human race, but one forgets, neglects and sacrifices with it many natural and inborn reflexes and does not know how much damage is done to the body (figs. 4, 5 and 6).

The next step in voice production concerns the laryngeal system as it is reached by the air column. The air column, produced and main-

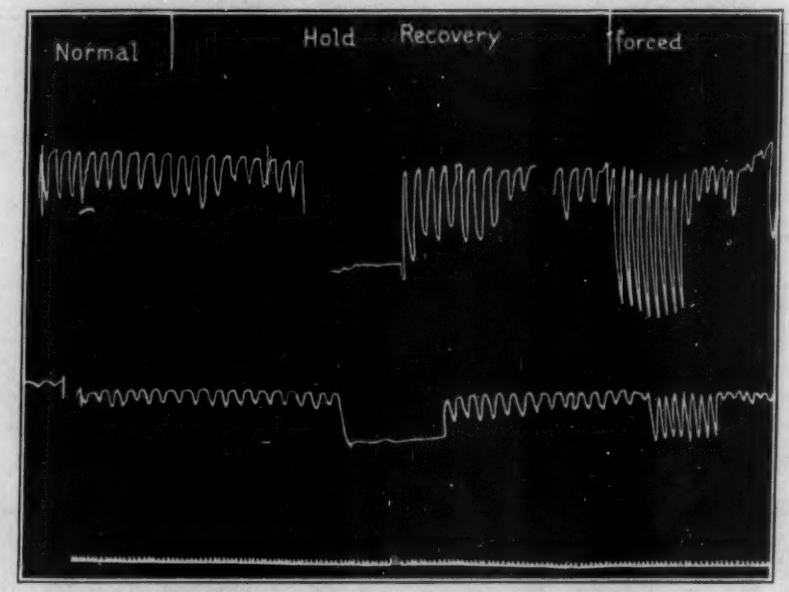

Fig. 3.—Pneumographic tracing of a girl aged 25 with vocal training.

tained by the respiratory muscles, mainly the diaphragm in conjunction with the vocal cords, also controls the pitch of the produced tone.[14] One can truthfully say one speaks and sings not with the larynx but primarily with the diaphragm and the abdominal muscles.

Anatomically, this system consists of a group of muscles working mainly on the thyroid, the cricothyroid and the arytenoid cartilages; these are called the intrinsic muscles of the larynx and include the posterior and lateral cricoarytenoid, the cricothyroid, the thyroarytenoid and the interarytenoid muscles. These muscles can be divided into a constrictor and a dilator group for closing and opening the glottis. The thyroarytenoid, the lateral cricoarytenoid and the interarytenoid muscles belong to the sphincteric group of the glottis and work on the muscular processes of the arytenoid cartilages, while the posterior cricoarytenoid

muscles serve as dilators of the glottis, also by acting on the muscular processes of the arytenoid cartilages in rotating them away from one another.

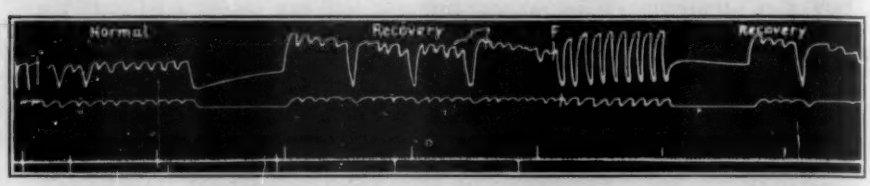

Fig. 4.—Pneumographic tracing of a man aged 45 without vocal training.

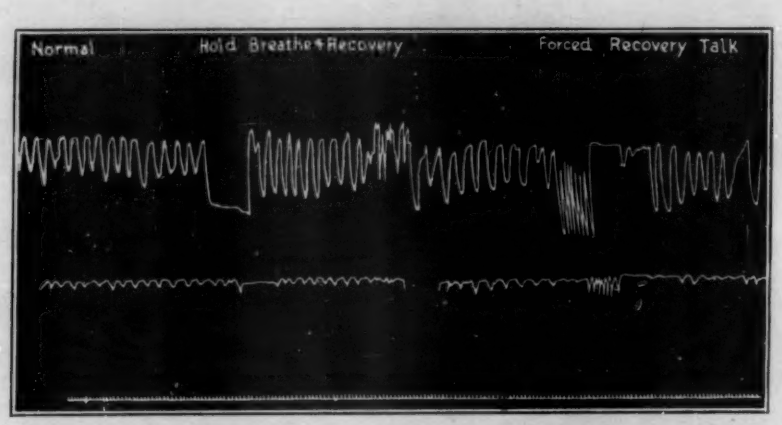

Fig. 5.—Pneumographic tracing of a woman aged 44 without vocal training.

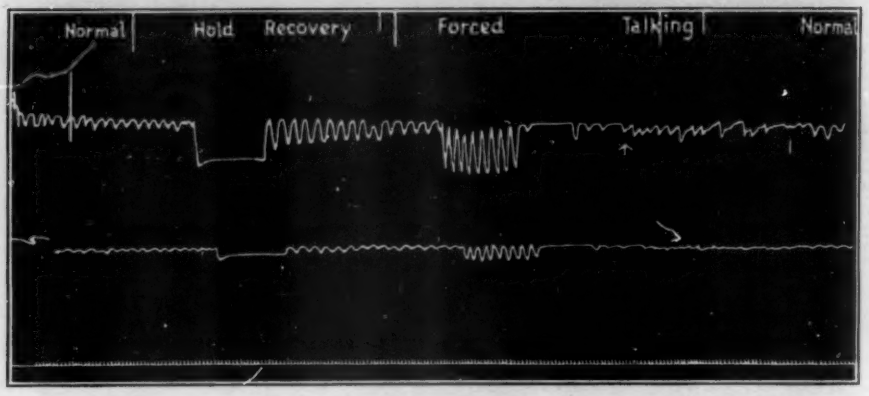

Fig. 6.—Pneumographic tracing of a woman aged 50 without vocal training.

There is normally an interplay between the air column and the intrinsic muscles, controlled by simple unconscious, and also conscious, reflexes. As already mentioned, the size and force of the air column

and the degree of contraction of the intrinsic laryngeal muscles determine the pitch of the produced tone. During phonation the membranous part of the vocal cord vibrates, while the arytenoid cartilages remain in firm apposition, held by the intrinsic muscles.

It is easily understood that these small and delicate muscles respond to very slight stimuli, e. g., to a balanced mechanism, which will be explained in more detail later.

The larynx, with its intrinsic mechanism, is a freely movable and very elastic structure. It is of special importance in phonation and one of the main factors in producing "functional hoarseness." The larynx as a whole is controlled by the so-called extrinsic muscles, consisting of depressors and elevators.[14] The elevators are the inferior constrictors of the pharynx, the longitudinal fibers of the pharynx proper, the thyrohyoid muscles and the stylopharyngeal muscles; the depressors are the sternothyroid muscles and the longitudinal fibers of the esophagus. These extrinsic muscles control the laryngeal movements in such an elastic and flexible manner that the caliber of the pharynx or the larynx is decreased or increased, depending on the need at the moment for deglutition or phonation. The interplay of intrinsic and extrinsic muscles is another important factor in the disturbance under discussion.

The Resonators.—Like any other musical instrument, the human vocal mechanism has chambers for resonation of the produced tone, thus enabling one to get a maximum amount of volume with a minimum amount of energy. The greatest of these resonating chambers is the bony thorax. Nature provided the female with shorter and thinner vocal cords, and it may be due to this difference that women overlook and neglect the presence of this thoracic resonance much more than men do. The deep and pleasant voice, so soothing to the nerves of the listener, is due to no special secret but to the free and ample use of the wide bony thorax as a resonating chamber, instead of squeezing the air column through a narrowed air passage.

Continuing upward through the air passages, the air reaches the oral cavity, which also belongs to the resonating part of the phonatory system. But in addition to the importance of the bony structures of the mouth and skull, one must consider the role of the muscles. The muscles of the tongue are in close relation to the hyoid bone, and through its connection with the larynx these muscles have great influence on the elevation and depression of the larynx. Furthermore, the muscles of the tongue and of the pharynx decrease or increase the resonating chambers. The muscles of the lower jaw, of the mouth itself and of the soft palate and the suprahyoid, as well as the infrahyoid, muscles are all concerned with phonation, partly by helping directly in the formation

of vowels and consonants and partly by decreasing and increasing the resonating chambers and pathways of the space above the larynx.

One can compare the whole phonatory system to an organ in the church. The bellows is the expiratory musculature; the keyboard is the larynx, with the vocal cords and muscles; the pipes and the resonating wall of the church, the resonating chambers, consisting of the cartilaginous and bony structures of the thorax, the oral cavity, the bones of the skull and the paranasal sinuses.

The different anatomic parts involved in this complex mechanism having been enumerated, it is understood that this mechanism works properly only if all parts act together in the right order, to allow smooth and frictionless motion and function. The slightest disorder disturbs this fine and delicate mechanism, and no force can bring about proper action unless the importance of the single parts and their combined action is understood.

PATHOLOGIC AND CLINICAL PICTURE OF FUNCTIONAL HOARSENESS

Etiology.—There are several probable factors in producing this pathologic condition.

1. Acute laryngitis, occurring at any age. The patient is not sick enough to stay in bed and spare the voice. But the hoarseness is a handicap in the daily work; in order to compete, the natural mechanism is abandoned; muscles which have nothing to do with phonation are used in order to force phonation. The patient is not conscious of this; the acute condition passes into the chronic phase.

2. Abuse of the phonatory mechanism from the start. A patient who has never been trained in correct phonation may acquire from childhood a wrong use of the mechanism and never recover.

3. Teaching in schools of wrong methods of respiration, with production of disturbances in the phonatory mechanism.

Symptoms and Signs.—Almost every patient with functional hoarseness presents one or more, maybe all, of the following symptoms: (1) frequent clearing of the throat; (2) excessive discharge; (3) tension in the structures of the neck, especially after long use of the voice; (4) possibly even difficulty in swallowing and the sensation of a lump; (5) easy fatigue; (6) irritability after long talking, and (7) psychic depression if the functional hoarseness is of long standing.

The signs are: (1) open glottis in phonation; (2) relaxed and irregularly shaped vocal cords; (3) redness of the cords, which may or may not be present; (4) thickening of the cords in most cases; (5) irritation of the mucous membrane; (6) tenseness of muscles of the neck, as compared with the other muscles, with spasticity of the sterno-

cleidomastoid muscle; (7) pulsation of vessels of the neck during phonation and visible contraction and dilatation of structures of the neck; (8) fixed lower jaw, with clenched teeth, and (9) speaking without use of the facial musculature.

These symptoms and signs do not constitute the whole picture. As in any other disease, there is a strong emotional and psychic component. Phonation and the ability to express one's thoughts and feelings are so important that a pathologic condition produces a definite psychic reaction. If one makes the effort to go into this clinical phase of the patient's case, one will find that the condition makes him irritable, nervous, retiring and frustrated; he avoids crowds because he cannot make himself heard above the loud voices and if he tries he has pain—burning and pricking sensations—so that he gradually gives up and an inferiority complex is formed.

Production of the Clinical Picture.—In the beginning of this article the different components of the condition were mentioned. The trouble starts with respiration. Even persons trained in correct diaphragmatic breathing, with strong movements of the abdominal musculature, do not always associate it with the rest of the phonatory mechanism. There is a wide space between the diaphragm and the larynx and head. The air column is not controlled. The air blows unrestricted and uncontrolled against the glottis. The intrinsic muscles and the cords have their limit in counteracting and responding to this uncontrolled and tremendous pressure from below; the cords relax, become thickened and inelastic and cannot close properly. Hoarseness starts because part of the air column rushes through the open glottis without being interrupted by the cords. Part of the air sets the relaxed vocal cords into vibration, and since these are no longer uniformly stretched and elastic, different tones, with variable amplitudes and frequencies of vibration, are produced. These waves interfere with each other, and a mixture of tones experienced as hoarseness is heard.

At the same time the patient starts to make up for the loss of voice by putting muscles and structures into action which ordinarily have only secondary functions in phonation. The depressors and elevators of the larynx, together with various external muscles of the neck, are used in order to get the necessary force of voice. These muscles exert pressure and pull on the larynx. The natural free mobility and elasticity of the larynx are lost. These muscles fix the position of the larynx and change the diameter in such way that the intrinsic muscles concerned with adduction and abduction of the cords are inhibited in their function. These intrinsic muscles are much too small to overcome the strong action of the extrinsic muscles.

Normally the vibrating air column is reenforced by the resonating chambers so that one is able to produce a maximum of tone with a minimum of energy. This is not so in cases of functional hoarseness. The air column, instead of reaching the oral cavity, the nasopharynx and the skull and paranasal sinuses, and also extending downward to fill the whole thoracic cavity, is compressed, cut off and distorted by the unnaturally exerted pressure and the decrease in the diameter of the pathways and chambers, and the patient uses the soft structures of the neck as the main resonators. By analogy, the tone is thrown not against a fixed sounding board, as in a musical instrument, which gives an even and uniform resonance, but against a soft sheet, which gives way when air is blown against it. There is no resistance, and ten times as much force has to be used, with a very poor result.

It is interesting to measure the differences in current energy used up by a person with a well trained voice and one with functional hoarseness. The pressure and pull constantly irritates the mucous membrane, causing frequent clearing of the throat of excessive secretion.

Last, but not least, is the rigidity of the facial structures. Most patients with this condition speak with an absolutely fixed mandible. In this way more pressure is exerted on the larynx, keeping this organ in a fixed position and so inhibiting its normal function. The facial muscles concerned with phonation are entirely inactive. The patients speak with almost closed lips and through the teeth and are difficult to understand. Here, again, they use up much more energy than is necessary in order to produce the desired phonation. The tongue likewise plays an important part.

TREATMENT OF FUNCTIONAL HOARSENESS

In view of these factors, the treatment is evident, namely, development of the natural function and elimination of the disturbing factors. The patient must be made conscious of the simple reflex mechanisms. He must be taught how to use all the organs of phonation. It is a matter of reeducation.

Respiration.—Put the patient on a flat stretcher or bed. Place the hands flat in the region of the diaphragm and exert firm pressure. Have the patient take a deep breath, telling him to push your hands away while taking this breath. Watch carefully that he does not raise the shoulders or upper portion of the thorax in inspiration and that he takes a really deep breath. Then exert intermittent strong pressure on the so-filled diaphragmatic region, thus gradually forcing the air out of the lungs; the patient must realize by experience that the diaphragm acts like bellows. Repeat this as many times as necessary to make the mechanism well understood.

Control of Muscles of Respiration.—The patient takes a deep breath, which of course pushes the abdominal wall outward, owing to the change in intra-abdominal pressure. After the patient inspires so that the lungs are filled with air and the diaphragm descends tell him to contract the abdominal muscles by pulling the abdomen inward without allowing the air to escape. In this way the abdominal muscles get control over the diaphragmatic action in expiration. Now tell the patient to exert, by slight contraction of the abdominal muscles, gradual pressure on the diaphragm and to release part of the air. Have the patient relax the abdominal muscles partly, then contract them again, so pushing out gradually all the inspired air. Again, the patient has to experience this mechanism until he gets control over the breathing mechanism. The abdominal muscles must become strong and elastic and must react on the slightest stimulus from the patient. It is important that the patient understands and feels the connection between the inspired air and the action of the diaphragm and abdominal muscles and the expired air column at the oral aperture and realizes that one is dependent on the other.

Closure of the Open and Relaxed Glottis.—As has been said before, the position and action of the laryngeal structures are distorted in cases of functional hoarseness. The physician's duty is to reeducate and transform this faulty mechanism. This is done by putting the vocal cords and intrinsic laryngeal muscles under pressure from above and below—from below by taking a deep breath in the way already explained; from above by blowing the cheeks out so that the whole oral cavity is full of air. This air is forced between the thyroid cartilages and the arytenoid cartilages and acts like a buffer. The lips are pressed together, so that very little air can escape. By forcing the air between the laryngeal and the extrinsic structures (fig. 9) one eliminates all undesirable pressure and pull exerted on the larynx. This enables the intrinsic muscles to do their work and at the same time stretches the cords. The pressure from above and below (fig. 9) hinders the cords from bending in either direction; they have to stretch in the natural way. After the patient understands this mechanism fully, he is asked to produce a low tone with this double air column. Even the hoarsest voice will be able to do this and produce a clear tone. Roentgenograms (figs. 7, 8 and 9) taken at this point show the mechanism. The intrinsic mechanism acts in the normal way; the intrinsic muscles stretch the cords, and they approach each other; vibration is brought about; the glottis closes, and one hears a clear, pure, nonhoarse tone. Again, this must be repeated until fully understood.

The importance of this exercise lies in the fact that the air column produced in the beginning, when just blown without tone, as well as

afterward, when blown with a tone, concentrates at the tip of the lips when the cheeks are blown out. By holding a finger in front of the lips one can feel the escape of this concentrated air current and later experiences the tone, which is proof that the air column is carried uninterrupted from the diaphragm to the larynx and to the lips. One must make sure that there is no interruption between diaphragm and larynx

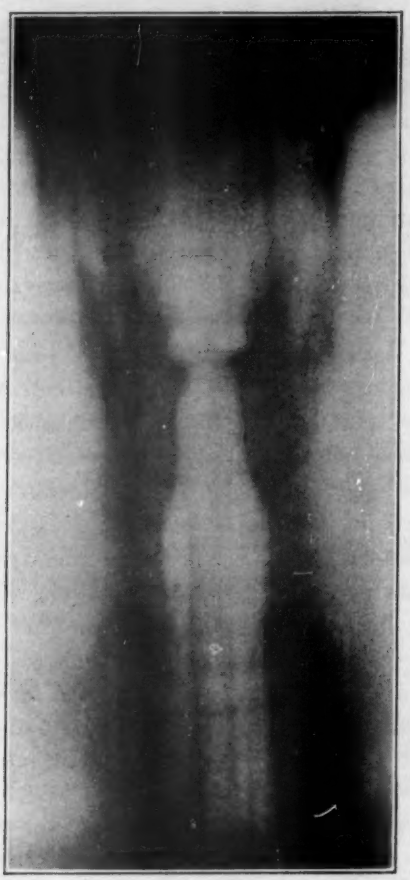

Fig. 7.—Glottis in inspiratory phase.

by exerting slight pressure on the upper portion of the abdomen while the patient blows the tone through the air-filled oral cavity and the closed lips. This pressure forces out more air, which can be felt at the tip of the lips. The patient must get the feeling of this and understand the action of this mechanism. Each higher pitch, each increase in the force of the tone, must be reached in this way, by passive or active pressure of the abdominal muscles, and not by any pressure in the laryngeal

region. The cords themselves are absolutely passive, as are all the surrounding structures. The intrinsic muscles respond to every increase or decrease in the infraglottic air column and change the length and tension of the cords.

The next step is to extend this exercise over the whole range of the patient's voice in order to massage the cords at all amplitudes and fre-

Fig. 8.—Glottis in phonatory phase in a case of functional hoarseness.

quencies of vibration. In passing from a lower to the next higher tone, slight pressure of the abdominal muscles forces an increased air column through the passages, changes the response at the vocal cords and appears at the oral aperture as a higher tone. Slight relaxation of the lower jaw with each higher tone is necessary. Soon one finds where the condition of hoarseness started, that is, at what pitch, for at that point

the patient shows a certain weakness and insecurity, a slight tremolo and impurity, of the tone. Thickening of the vocal cords inhibits normal elasticity and irregularity causes the tremolo and impurity. The affected region has to be emphasized in the exercise until the defects are overcome, and in repeating the laryngeal examination one will find that the

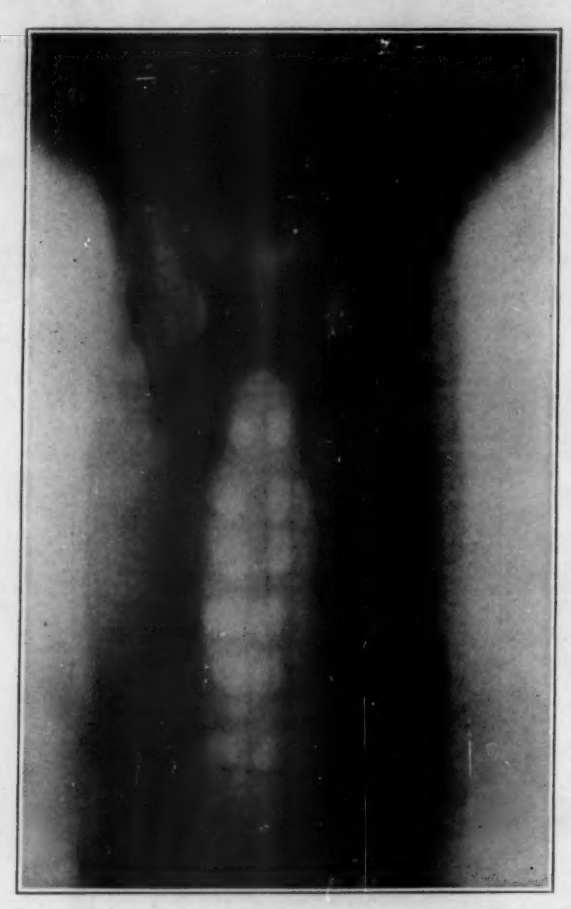

Fig. 9.—Glottis under supraglottic and infraglottic air pressure.

vocal cords regain their elasticity and tension and that the glottis closes normally in phonation.

Not only does this exercise close the glottis and reeducate the cords, but soon the patient feels great improvement in the previously experienced tension. The structures of the neck begin to relax. The previous spasmodic contraction of the muscles disappears; the irritation of the mucous membrane subsides; there is no excessive secretion and no fre-

quent clearing of the throat. The nervousness and irritability disappear. The fault is repaired, but the normal phonatory mechanism has to be built up in leading the voice into the natural channels.

The next step is placement of the voice. There are several ways to make the patient feel the location of resonance.

PLACEMENT OF THE VOICE

When there is foreign material or mucus in the nasal passages, one gets rid of it by blowing hard through the nasopharynx. The same path should be taken by the tone. To accomplish this have the patient blow through the nose and control at the nares the outcoming stream of air. This has to be practiced several times until the patient actually experiences the passage of the air column which is initiated at the diaphragm and supported by it and the abdominal musculature. In blowing out, the passage of this air column should be combined with the production of a low tone, convenient for the particular voice. The vibration of this tone can be felt at the root of the nose, and it is important that the patient feels it in the bony structures in front of the nasopharynx. At the place where

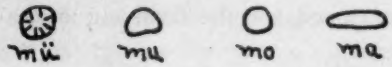

Fig. 10.—Position of lips for vowel phonation.

the patient feels this single tone, each spoken word or singing tone must be placed. In the beginning it is best to start each tone with the little blow through the air passages, in order to give the patient a definite feeling about the location of the voice. Have the patient say, or better still sing, "mü" and later "ma," using this mechanism. Watch that the vowels are formed distinctly at the tip of the lips. In this way the facial, and especially the oral, musculature will be trained. The whole air column appearing at the oral aperture has to be transformed into spoken or singing tones, and therefore the air column has to be concentrated. If wild air escapes through together with the tone, an impure and hoarse tone will be heard. The lips have to be formed for the various vowels, so that everybody can read the intended vowel from the lips without hearing it (fig. 10).

Of further importance is the fact that no special effort or strength should be used to produce these tones. The structures of the neck must be relaxed all the time, and only the vibrations of the air column should be felt all along the air passages, from the larynx upward to the nasopharynx. Again and again it must be emphasized that the connection between the diaphragm and the nasopharynx, respectively, and the oral cavity must be uninterrupted. By placing the tone in the nasopharynx

advantage is taken of all the resonating chambers in the skull, the paranasal sinuses and the oral cavity. The start of the tone is experienced behind the root of the nose, but the vibrations are transmitted and reenforced by all the aforementioned bony structures.

When the patient understands the mechanism of the placement of the voice as combined with the respiratory mechanism, sequences of tones should be tried, the tone starting always in the nasopharynx and being supported by the air column. By slight abdominal pressure and slight dropping of the lower jaw the next higher tone will be easily obtained. Relaxation and absolute flexibility of the mandible are most important. The jaws must be relaxed, and likewise the tongue. All pressure and contraction must be eliminated from the neck and transferred to the diaphragmatic region.

After the mechanism of placement of the voice is fully understood, the patient should start to read in the same way, placing each word in the nasopharynx and extending the volume down into the thoracic cavity. The vibrations of the spoken and the singing voice should be felt over the upper portion of the thorax. By training and cultivating especially the low tones, which are mostly used in speaking, more and more chest resonance can be developed, and the shrill and unpleasant pitch gradually is lost.

SUMMARY

It is the purpose of this paper to outline and describe the condition of functional hoarseness, its diagnosis and treatment. Functional hoarseness is a symptom complex of the laryngeal and surrounding structures, with the outstanding symptom of hoarseness. In order to treat this disturbance the patient must be taught (1) the mechanism of respiration; (2) relaxation of the lower jaw and tongue; (3) massage of the vocal cords by supraglottic and infraglottic pressure with an air column; (4) placement of the voice; (5) use of facial musculature in the formation of vowels, and (6) cultivation of all resonating chambers, with emphasis on the thoracic.

In the preparation of this paper I have frequently sought the advice of staff members of the Woman's Medical College of Pennsylvania: namely, Dr. Emily Lois Van Loon, Professor of Otolaryngology; Dr. Esther Greisheimer, Professor of Physiology; Dr. Sarah I. Morris, Professor of Preventive Medicine; Dr. Jacob Vastine, Clinical Professor of Radiology; Dr. Hartwig Kuhlenbeck, Professor of Anatomy, and Dr. Pauline Coonel, Instructor in Clinical Otolaryngology.

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FREQUENCY OF HOARSENESS DUE TO PHONATION WITH THE THYROARYTENOID LIPS

JACKSON'S DYSPHONIA PLICAE VENTRICULARIS

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The part played by the supraglottic area, the so-called laryngeal aditus, in voice and phonopathic conditions has long been known to be considerable. This had been pointed out long before the fundamental concepts of vocal function were upset by Cotton,[1] in 1940, when he verified experimentally the postulates of Husson and Tarneaud.[2] In 1902 Scripture [3] suggested that the false vocal cords might well have a definite function. Such a function in the production of voice was defined in 1915 by Muckey.[4] He formulated a law, which has since proved to be true, namely, that correct voice production may be summed up as noninterference with (1) the action of the vocal cords and (2) the opening of the tube that leads out through the pharynx, the buccal cavity and the lips. Muckey went on to say that as a strong fundamental tone is important in the production of volume and quality, it may readily be understood that interference from the false cords is detrimental to both qualities. The pulling in of these soft parts makes the cords heavier. Such interference is a fruitful source of faulty intonation. The so-called nasal twang is due entirely to this factor. In fact, any roughness or harshness in the tone is apt to be the stigma of interference from the false cords. The twang was caused only by interference from these cords of a definite pattern was verified by Russell.[5] This must be differentiated from Cotton's disease, the so-called whang, which I [6] correct by stimulating the gag reflex with a probe.

From the Speech Clinic of the Oklahoma College of Agriculture and Mechanical Engineering.

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3. Scripture, E. W.: Elements of Experimental Phonetics, New York, Charles Scribner's Sons, 1902, p. 265.

4. Muckey, F. S.: The Natural Method of Voice Production in Speech and Song, New York, Charles Scribner's Sons, 1915, p. 73.

5. Russell, G. O.: Speech and Voice, New York, The Macmillan Company, 1931, pp. 236-242.

6. Voelker, C. H.: Phoniatry in Dysphonia Ventricularis, Ann. Otol., Rhin. & Laryng. **44**:471, 1935.

Negus [7] described how the false cords close and traced their action phylogenetically. He further described certain of their actions in man and in the higher apes. They act like a heart valve, in that air pressure in the appendix of the larynx causes them to balloon up in certain types of tussis and thoracic fixation.

Russell,[8] using his laryngeal periscope, studied the physiologic action of the false cords. He pointed out that almost every one, with the exception of Muckey, had expressed doubt as to whether they had any muscular function and that some had even said that they were not subject to muscular constriction. With his methods, he found their violent contraction and sudden explosive separation strikingly manifest in tussis. According to the old theory, long since disproved by Wyllie,[8] when a cough or sneeze became necessary, the glottal lips were brought tightly together and the epiglottis dropped down to shut off the "Sunday" throat, and then both together were suddenly forced up, with violent explosion. Russell's experiments indicated that the present day concept of the primary function of the epiglottis is one of vowel quality and that the epiglottis is not a functional part of the cough. Farnsworth's [9] high speed motion pictures of the vocal cords, taken at 4,000 frames per second, clearly showed that only the pulvinar of the epiglottis was active in the sphincter and that only after the pneumatic explosion was entirely past did the epiglottis flutter in the current of air. In Farnsworth's pictures there is verification of Russell's contention that the false cords, acting in unison with the pulvinar of the epiglottis and the prominences of Santorini and Wrisberg, actually are capable of tight constriction, so tight that not the slightest strip of the white true vocal lips underneath is visible.

Although the physiologic aspects are well known, the myologic possibilities have yet to be described anatomically. As I [6] pointed out, these ventricular bends are found to produce a vicarious voice. This voice can be made more pleasant by training, so pleasant that it is possible to sing with a false cord voice. On the other hand, it can be made more growling by training. Usually the voice without training is so hoarse that it is phonopathic. It is what is commonly called a muttering voice. The false cords also act to supply the lower pitch in "squealing" diplophonia but probably have little function in Paget's "singing" diplophonia.

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7. Negus, V. E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1931, pp. 442-466.

8. Wyllie, J.: The Disorders of Speech, Edinburgh, Oliver & Boyd, 1894, p. 463.

9. Farnsworth, D. W.: High-Speed Motion Pictures of the Human Vocal Cords, Bell Lab. Rec. **18**:203, 1940.

Attention should be called to the belief that a consonant can be the result of action of the ventricular musculature. Linguistically, the glottal stop may be of this origin. Some explosives are formed by the buccal occlusion plus a glottal stop. The glottal stop substitutes for an explosive. The treatment of certain types of nasality, cleft palate postoperative speech and short palate speech is based on the use of the ventricular bands simultaneously with the buccal gesture in the articulation of explosives.[10]

The discovery of substitute phonation with the ventricular bands was made independently by workers in several countries within the last ten years. It was described by Flatau[11] (Germany, 1928); Heymann[12] (Germany, 1931); Negus[7] (England, 1931); Réthi[13] (Hungary, 1933); Nadoleczny[14] (Germany, 1934); Voelker[6] (United States, 1935); Jackson and Jackson[15] (United States, 1935); Hayashi[16] (Japan, 1936), and Tarneaud[17] (France, 1937). These investigators have all agreed that good results in treatment are not accomplished by ordinary singing exercises, but are obtained only from diagnostic phoniatric procedures. I[6] have completely ruled out tension exercises, which were aimed at bringing the true cords together. This is reasonable, since the closure of the false cords occurs with any high degree of tension or fear. Thus, I have recommended an opposite type of therapeutic technic,[6] an incipient pandiculation, which is used as a sort of speaking stance. Since this development the bronchoscopic position has been used successfully. The patient gains auditory control of quality while in this position and then, in gradual stages, maintaining this auditory control, he lowers his head to its normal position. Clinical experiments have conclusively proved that another law of voice production should be stated, namely, that voice produced while the subject

10. Voelker, C. H.: Therapeutic Technique for Staphylolalia, Arch. Otolaryng. **21**:94 (Jan.) 1935.

11. Flatau, T.: Zur Klinik der Taschenbandstimme, Monatschr. f. Ohrenh. **62**:791, 1928.

12. Heymann, O.: Taschenfaltenstimme auf organischer Grundlage, Ztschr. f. Laryng. **41**:500, 1931.

13. Réthi, A.: La voix des bandes ventriculaires et la musculature du pharynx, Ann. d'oto-laryng., 1933, p. 556.

14. Nadoleczny, M.: Pathologische Phonationsmechanismen, in Festschrift, Ino Kubo zu seinen 60. Geburtstag (26. Dezember 1934) von seinen ausländischen Freunden gewidmet, Tokyo, The Herald Press, Inc., 1934.

15. Jackson, C., and Jackson, C. L.: Dysphonia Plicae Ventricularis: Phonation with Ventricular Bands, Arch. Otolaryng. **21**:157 (Feb.) 1935.

16. Hayashi, Y.: Ueber die Ersatzstimme, Oto-rhino-laryng. **7**:641, 1936.

17. Tarneaud, J.: Affections vocales et bandes ventriculaires, Ann. d'oto-laryng., 1937, p. 314.

is in what physical educators call the A posture is both natural and efficient. Grab the patient by the hair, stretch him up and command "Say, Ah!," and if it is all done quickly enough an efficient and natural voice will be heard. As West [18] has in part indicated, a short cut for assuming the A posture is possible. It consists of a clinical exercise in which the patient practices phonation while maintaining the greatest possible distance between the ears and the shoulder and between the hyoid bone and the laryngeal prominence. Another method of attack is to give the patient exercises in maintaining the thyroid incisure as close to the clavicle as possible.

Exact clinical methods for examining for substitute voice have been completely and adequately described by Jackson and Jackson,[15] Hayashi [16] and Tarneaud.[17] But it might be added that even in patients in whom the actual phenomenon is not observed in the mirror, evidence of the presence of chronic, and sometimes acute, hoarseness can be clearly seen in the texture and coloring of the mucous membranes of the ventricular bands. The globular, ciliated, pseudostriated, columnar epithelium will be modified, through use, into the stratified squamous type. Furthermore, this muttering voice can be detected by ear, since it sounds distinctly different from other types of hoarseness. A stethoscope will help distinguish its peculiar quality. However, even if a clinician has a developed phonetician's ear, a visual examination is always recommended.

In phonation with the ventricular bands the voice not only sounds hoarse but is much deeper than the natural voice. Oscillographic studies indicate pitches of the order of 50 or 60 cycles, which indicates that the key is between that of the natural voice and that of a laryngectomee. The voice is apt to sound rough and crackling. The more chronic the use of this vicarious sound source, the more unpleasant the sound, but, in contrast to this, the more acute the hoarseness and the more frequently ventricular phonation is used, the smoother the voice. In fact, after excision of the vocal cords, very pleasant ventricular phonation can be developed. Ventricular voice is apt to be weak, and it is this weakness that causes the patient to seek attention of the physician or the speech clinic. The complaint of phonasthenia seems strange, since ventricular action is related to the explosive physiologic functions in which the sound, if it is produced at all, as in coughing, sneezing, thoracic fixation or grunting, has considerable volume and carrying power. In fact, an actor was once trained to imitate a lion's roar with the ventricular bands so that he could save his vocal cord voice through the long rehearsals and extended performances. That

18. West, R. W.: The Function of the Speech Pathologists in Studying Cases of Dysphonia, J. Speech Disorders **3**:81, 1938.

roar was completely adequate to fill the auditorium and even had the lion's ventriloquial effect in that it seemed to come from everywhere. Patients coming to clinics for ventricular hoarseness or phonasthenia regularly complain of clearing mucus from the throat and of hacking and coughing.

One patient complained of dropping his voice at the end of sentences, and it was found that he did not lower his vocal cord pitch but actually stopped using his vocal cords at the end of the sentence and substituted for them a ventricular vibration. An actor, with an excellent stage voice, complained of hoarseness only in conversation. It was found that in intimate and quiet conversation he used a ventricular voice to "save for his art" his stage voice. A youth was criticized by his parents for having a high and squeaky voice and acquired ventricular phonia in order to lower his voice. Thus, instead of lowering his voice to a normal pitch, of perhaps 150 cycles, he lowered it to one of between 48 and 57 cycles. A similar case was found in which a man 31 years old, who had a deaf wife, became self conscious about his yelling and outside his home developed phonation with the ventricular bands to subdue his voice. A college student raised the pitch of his voice to read aloud or to recite but used a ventricular tone in conversation. Sometimes it is found in careless conversation only. A 5 year old boy was kidded by his playmates for having a high voice, and he lowered it by acquiring a ventricular voice. An 18 year old youth, with a eunuchoid quality, substituted ventricular phonation for his weak and strident vocal cord voice and thought his new hoarse voice gave the impression of masculine virility.

Patients have been examined whose hoarseness seemed to have been related to psychoneuropathy. This should have been anticipated, since it was demonstrated by Kenyon [19] that the mechanism of nervous manifestations, that is, the mechanism for producing the low hydrogen ion concentration, the pallor, the blood concentration in the cranial region, the gasping, etc., is simply "holding the breath" with the ventricular bands. Wedberg [20] described this in the case of his being afraid when shut in a dark closet. All he had to do, since he was an adult and was not afraid any more, was to release his ventricular sphincter and breathe, and immediately his fear and perspiration stopped. It seemed that since he had been afraid as a child, he unconsciously held his breath. He brought about this reaction by repeating his childhood punishment of being shut in a dark closet. He locked himself in and threw down the key. Immediately he stopped breathing, got down on

19. Kenyon, E. L.: The Etiology of Stammering: An Examination of Certain Recent Studies, with a Glance into the Future, J. Speech Disorders **6**:1, 1941.

20. Wedberg, C.: The Stutterer Speaks, Boston, Expression Co., 1937, p. 124.

his hands and knees in the darkness and groped for the key. He stopped, collected his wits and tried to analyze objectively why his heart beat so hard. He found he was not breathing. He started to breathe deeply, and then all fear response left him.

It might be expected that nervous patients would tend to start out with a ventricular voice and, as they gained in poise, would clear the throat and resume speech with a clear vocal cord tone. A 14 year old boy, who had lived in an oppressive social milieu, demonstrated his meekness overtly by having very little verbal output and, when he did talk, using an athymic ventricular phonation. On the other hand, in examining a college student for a pathologic cynical attitude it was found that a part of his overt cynicism was expressed as ventricular phonia.

Adolescents who are exuberant but puerile, as a result of a lag in social maturity, almost regularly demonstrate a special configuration of vocal qualities, which include the ventricular timber. There is, in these cases, a lack of syntactic completeness in sentence structure, tanyphonia (thinness of tone) and habitual functional staphylophonia (talking through the nose),[10] together with intermittent ventricular phonia.

Ventricular phonia has been seen in patients who have had labial malformations, sluggish lips, thick lips, dental separation, malocclusion and the like, as well as in former stutterers, in neurotic clutterers and in persons with a nervous, rapid or quivering speaking voice. It has also been seen in bilingual persons, and in one of these polyglots it appeared only in the secondary tongues. It has also appeared together with immobility of the upper lip and unilateral flaccid facial (paralytic) dysarthria.

Jackson and Jackson [21] stated:

> The frequency of hoarseness due to phonation by the ventricular bands is impossible to determine by general statistics because the condition evidently has not been recognized by laryngologists. Our own records of cases have not been regularly cross-indexed for this condition, but a fair estimate is that in about 4 per cent of the cases of hoarseness encountered the dysphonia is due to this vicarious assumption of phonatory duty by the ventricular bands.

PRESENT STUDY

In a speech clinic the number of these cases of functional disturbance in iatrogenic practice could not be determined; so a study was set up in which not the frequency in a laryngologist's practice would be determined but, instead, the frequency in the general population. Owing to many situational factors, the study was limited to 1,871 males in late

21. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, p. 117.

adolescence. These young men were given a routine speech examination, and they tried their best to impress the examiner. A part of this examination was devoted specifically to ascertaining the use of the ventricular bands. It is believed that this sample, although monosexual, is adequate enough to be reliable.

This examination of approximately 2,000 males was conducted so as to sift out for special study those who demonstrated ventricular phonia. Exactly 113 were found, an incidence of 60 per thousand, or 6 per cent. However, in only 1.2 per cent was the hoarseness acute. In the other 4.8 per cent the hoarseness proved to be chronic in the phoniatric sense of intermittent. Careful study of the subjects with acute hoarseness finally indicated that even in a case of acute hoarseness a vocal cord voice might accidentally be produced under exceptional conditions. However, in the early days of treating patients with this condition, before efficient measures had been discovered, 1 patient was studied five days a week for over a year without accidentally producing a vocal cord tone. In addition to the 6 per cent (representing both acute and chronic hoarseness), it might logically be supposed that almost every one uses the ventricular bands for voice at some time.

Although it was not possible to compare iatrogenic hoarseness (ordinary medical and inflammatory types) with ventricular hoarseness, since, unfortunately, the records were not so kept, it was possible to compare the frequency of the vicarious ventricular voice with the frequency of other dysphonias. Of all the acute phonopathies, 16.5 per cent were found to be due to phonation with the ventricular bands.

Phoniatric complications occurred in approximately half the patients (52.2 per cent). The acute form was more likely to be complicated by other phonopathies than was the chronic. Complications occurred in 78.3 per cent of cases of the acute form and in 45.6 per cent of cases of the chronic form.

About half the patients had no complication, but a third (33.6 per cent) had logopedic troubles with articulation. These dyslalias are principally rhinolalia aperta, 12.4 per cent; parasigmatalalia, 7.0 per cent, and what the French call *zezaycism* (parażhetalalia), 5.3 per cent. Next in frequency were the complications of a vocal nature. Two patients had glottal complications. About half the time they used pneumophonia (breathiness) and the rest a ventricular rattling voice (muttering). These two types of trachyphonia (general term for hoarseness) have similar laryngeal postures and similar treatments. Dysphonias complicated 20.3 per cent of the cases of ventricular phonia. The usual complication was thin voice, in 8.8 per cent, and twang, in 6.2 per cent. Intonation was disturbed in 11.5 per cent of the cases, and this was mostly concerned with duration, in 8.8 per cent. Psycho-

neurotic vocal complications occurred in 3.5 per cent of the cases of ventricular phonia. Spasmophemia (stuttering) was three times as frequent among persons with hoarse voice as in the general population. This is substantially four times more than the probable statistical error and is therefore significant.

CONCLUSION

An investigation of the frequency of vicarious phonation with the ventricular bands was made through a survey of almost 2,000 male adolescents. The occurrence of this type of functional hoarseness was found to be 60 per thousand. However, in only 12 per thousand was there complaint of hoarseness. The other 48 per thousand were conscious of their voices when they tried to speak well but could not be said to complain of hoarseness in the usual medical sense. It is believed that perhaps every one uses the superior thryoarytenoid folds in speaking at some time in his life.

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PREVENTION OF DEATH IN STATUS ASTHMATICUS

VALUE OF BRONCHOSCOPY

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Six of the 7 patients who died of status asthmaticus at the Mount Sinai Hospital and on whom autopsy was performed in the past fifteen years died from blocking of the tracheobronchial airway by excessive outpouring of secretion.

A review of the literature revealed that it was only the exceptional case in which at autopsy a significant amount of obstruction by secretion in the tracheobronchial tree was not recorded.

The causative relation of a large amount of secretion in the bronchial tree to asphyxial death was more obvious in some cases than in others, but in the majority the presence of this secretion was the most important factor.

In many instances death could have been averted by bronchoscopic removal of the obstructing secretion.

The trachea and bronchi down to the fourth and fifth order may be cleared of secretion under direct vision by suction through the bronchoscope. Because of the tenacious character of the mucus, plugs obstructing the lumens of the smaller bronchi may also be removed. The clearing of the larger bronchi, in addition to affording an open airway, provides a reservoir into which the secretion from the smaller branches may be poured. If these larger branches are filled with mucus, there may be a damming back into the smaller rami and the active respiratory tissue.

The use of bronchoscopic procedures in the treatment of the less acute types of asthma is not new. Many patients subject to episodes of moderately severe asthma have been treated bronchoscopically and have obtained relief. There have appeared in the literature reports by Lukens,[1] Clerf,[2] Andrews[3] and others. These authors found the

1. Lukens, R. M.: Bronchoscopy in the Treatment of Asthma, Laryngoscope **35**:227-234 (March) 1925.

2. Clerf, L. M.: The Present Status of Bronchoscopy in Bronchial Asthma, Ann. Int. Med. **9**:1050-1056 (Feb.) 1936.

3. Andrews, A. H., Jr.: Bronchoscopy in Bronchial Asthma, Illinois M. J. **73**:25-31 (Jan.) 1938.

majority of the patients benefited by bronchoscopic treatment, the most marked relief occurring in those who had the greatest amount of secretion in their bronchi.

Bronchoscopic relief can serve as a life-saving measure for patients suffering from progressive respiratory obstruction and gradually supervening asphyxia due to status asthmaticus. Aspiration of the obstructing secretion, by increasing the effective airway, may be sufficient to allow adequate aeration of the lungs, thus tiding the patient over the critical period. In 6 of the 7 reported cases, if bronchoscopic intervention had not been withheld too long and the mucus had been removed before the asphyxial stage had become too far advanced, death might have been averted.

In the series of cases reported, there have been included only those in which death was directly attributable to status asthmaticus. Those in which death was due to supervening pneumonia or to congestive heart failure, for example, have been excluded.

REPORT OF CASES

CASE 1.—A 23 year old woman had had episodes of asthma for six months. She was in status asthmaticus for two days before her death.

Autopsy.—There were no areas of consolidation. The trachea was injected. The bronchi throughout both lungs were filled with very thick bloody mucus. Microscopic examination showed the bronchi to be dilated and filled with mucus, mononuclear cells and exfoliated cells. The mucosa was hyperplastic and the musculature hypertrophic. Numerous eosinophils were seen in the thickened walls. Alveoli near a bronchus were occasionally filled with mucus.

CASE 2.—A 67 year old man had been known to have asthma for about five years. He entered in status asthmaticus and died of asphyxia in spite of treatment.

Autopsy.—The lungs were voluminous, and cut sections of the two organs appeared similar. The branches of the bronchial tree were prominent and contained abundant thick mucus. The bronchi were intensely reddened and swollen and contained a large amount of mucopus. The terminal bronchioles showed this extensively. Microscopically, the lungs showed chronic productive and exudative inflammation of the bronchial tree, with round cell infiltration of the walls and hyalinization of the basement membranes.

CASE 3.—A 62 year old white woman had had asthma for five years before admission. She died in status asthmaticus.

Autopsy.—The lungs were strikingly voluminous. The bronchi were injected and contained viscid mucoid material.

CASE 4.—A 30 year old white woman had had asthmatic episodes for nine months. While in the hospital, she coughed constantly, expectorating large quantities of thick sputum, which at times formed almost complete bronchial casts. She died in typical status asthmaticus.

Autopsy.—The pulmonary parenchyma pitted on pressure and was extremely crepitant. The cut surface varied from bright red to pink. From all the small bronchi there exuded thick mucoid material. When the bronchi were opened and

traced to their larger branches, it was found that all the small and the large bronchi were completely filled with this same material, which formed casts of the lumens. The trachea was slightly reddened and likewise contained the exudate. Microscopic examination showed all the bronchi to be much dilated. Their lumens were almost all completely filled with matter which in places was pale, almost homogeneous and poor in cells. In other areas the exudate was largely cellular. This was composed of round cells, plasma cells, polymorphonuclear cells and eosinophils. Mucus threads and Charcot-Leyden crystals were seen in abundance.

CASE 5.—A 38 year old white woman had had asthma for eleven years. She died in status asthmaticus.

Autopsy.—The lungs weighed 980 Gm. They were moderately voluminous, hypercrepitant and fairly firm. From the small bronchioles a sticky white mucoid material could be lifted in long strings. The bronchi, from the bifurcation to the smallest branches, contained this tenacious viscid whitish mucus. There were no bronchial casts. The trachea was not unusual. A section of lung showed active mucus secretion within the larger bronchi.

CASE 6.—A 43 year old white woman had had asthma for three and one-half years. She died in status asthmaticus.

Autopsy.—There were acute and chronic tracheobronchitis with obstruction of the large and the small bronchi of all lobes by mucus. There was marked emphysema.

CASE 7.—A 54 year old white man had had asthma for three months. He died in status asthmaticus.

Autopsy.—The lungs were hypercrepitant throughout and at the periphery the lobes could be compressed to paper thinness. The cut surface of the lung showed a distinctly spongy stricture. There were no areas of consolidation. Some of the smaller bronchi contained mucus plugs. The trachea and large bronchi were clear. Microscopic examination showed no change in the pleura; in one section a large bronchus contained a cast composed of whorled layers of pink and light blue-staining material and enmeshing a large number of eosinophils and lymphocytes. The wall of the bronchus showed a marked cellular infiltration, consisting of eosinophils, plasma cells and lymphocytes. The bronchial lining epithelium showed evidence of active secretion. There was hypertrophy of the smooth musculature of the bronchial wall. The parenchyma showed empty, markedly distended alveoli, with rupture of the alveolar walls. The respiratory bronchioles were distinctly distended.

Comment.—In this case, obstruction due to bronchial secretion was probably of secondary importance.

REVIEW OF REPORTED CASES

We have reviewed the more recent reports in the literature of death due to status asthmaticus and have noted that the outstanding finding in the great majority of cases was partial or complete obstruction of the tracheobronchial tree by thick, viscid, tenacious mucoid secretion. In this review, also, only cases in which death was due to status asthmaticus itself have been included.

Recorded Cases in Which Death Was Due to Status Asthmaticus

Author	Case	Findings
Lemierre, Léon-Kindberg and Lévesque [4]	..	Bronchi plugged with a castlike formation containing many eosinophils and Charcot-Leyden crystals
Murphy and Case [5]	..	Large amount of tenacious, stringy mucus in the bronchi and their branches. Death attributed to asphyxia from bronchial obstruction
Michael and Rowe [6]	1	Fairly large gelatinous plugs obstructing lumens of many of the middle-sized and intermediate bronchi
	2	Entire right bronchial tree from the bifurcation of the trachea was obstructed. On left side all medium-sized bronchi showed almost complete occlusion
Loman [7] (in discussion of report by Michael and Rowe)	..	Plugs were found which had become canalized, much as a thrombus in the vascular tree may. These canals, in turn, were filled with very tenacious mucus, probably of recent origin. Mechanical obstruction was an important factor in the fatal termination
Fisher and Beck [8]	..	Bronchi and bronchioles were completely plugged with tenacious mucus
Wiseman [9]	..	Bronchi throughout both lungs were filled with thick tenacious mucus
Huber and Koessler [10] (Leyden's case)	..	Many small bronchi were almost completely occluded by a layer of amorphous material, which was adherent to the epithelium
(Berkart's case)	..	Right main bronchus almost completely occluded by dark brown cylindric branching masses. Smaller bronchi dilated and some partially obstructed by exudate
	1	Lumens of all bronchi contained only a small amount of granular precipitate, in which were fragments of desquamated epithelium
	2	Trachea and main bronchi contained a small amount of tenacious mucopurulent exudate. A few medium-sized and smaller bronchi were patent, but most were almost or completely occluded by grayish tenacious exudate and their walls thickened
	3	Middle-sized and smaller bronchi of both lungs were almost completely occluded by screw-shaped clots, which could be pulled out with difficulty. In larger tubes these clots were looser and several centimeters in length
	4	Many smaller bronchi were filled with shiny and often greenish-colored plugs
	5	All smaller and most larger bronchi completely occluded by tenacious yellowish white mucus
	6	Trachea and large bronchi contained purulent secretion. Many bronchi were occluded by masses of mucus
	7	Most smaller bronchi in upper and lower lobes were filled with a tenacious material. Larger bronchi contained a more fluid yellowish gray material

4. Lemierre, A.; Léon-Kindberg, M., and Lévesque, J.: Asthme et bronchite mucomembraneuse, Presse méd. **31**:613 (July 14) 1923.

5. Murphy, J. A., and Case, E. A.: Sudden Death from Bronchial Asthma, J. Allergy **1**:434-437 (July) 1930.

6. Michael, P. P., and Rowe, A. H.: Pathology of Two Fatal Cases of Bronchial Asthma, J. Allergy **6**:150-174 (Jan.) 1935.

7. Loman, in discussion on Michael and Rowe.[6]

8. Fisher, A. M., and Beck, J. P.: Death in Asthma: Report of Case with Autopsy, J. Allergy **2**:149-160 (March) 1931.

9. Wiseman, J. R.: Report of Case of Status Asthmaticus with Autopsy, Ann. Int. Med. **11**:212-221 (July) 1937.

10. Huber, J. L., and Koessler, K. K.: The Pathology of Bronchial Asthma, Arch. Int. Med. **30**:689-760 (Dec.) 1922.

Recorded Cases in Which Death Was Due to Status Asthmaticus—Continued

Author	Case	Findings
	8	Left chief bronchus and branches were almost completely occluded by a tenacious exudate. Middle-sized bronchi contained tenacious mucus, and finer branches contained a more fluid yellowish turbid exudate. Trachea contained an adherent mass of mucus which extended into right main bronchus
	9	Bronchi up to smaller branches were filled with tough yellowish white masses
MacDonald [11]	1	Smaller bronchi contained mucous plugs. Majority of intermediate bronchi showed laminated and stratified mucus filling lumens
	2	Lungs revealed striking emphysema. Many bronchi contained plugs of mucoid material
	3	Mucous glands were not prominent. Few of larger bronchi contained granular mucinous material in small amounts, with desquamated epithelium
	4	In some areas bronchial lumens were filled with mucoid homogeneous masses. Mucous glands were distinctly hypertrophic
Bubert and Warner [12]	1	Complete stenosis had occurred as result of extreme spasm during profuse outpouring of mucus
	2	Trachea and large bronchi completely filled
Cohen and Rudolph [13]	1 and 2	At autopsy, entire bronchial tree was filled with mucous exudate so tenacious that it could hardly be pulled away from the bronchial wall
Kountz and Alexander [14]	1	Large amount of exudate in bronchi
	2	Small bronchi contained large amount of exudate
	3	Small bronchi contained large amount of exudate
	4	Small bronchi filled with thick tenacious sputum
Cabot case record [15]	..	All bronchi filled with tenacious mucus
Steinberg and Figley [16]	..	Bronchioles and medium-sized and small bronchi filled with tenacious white mucus

The observations at autopsy in the 7 cases in which autopsy was performed at Mount Sinai Hospital and those reported in the literature are so striking that it is felt that bronchoscopic suction should be performed on patients who, in spite of all therapy, continue to go downhill. It is believed that if increasing dyspnea, deepening cyanosis and progressive weakness lead to the point where semistupor supervenes, at least at this point, if not before, bronchoscopy should be resorted to.

Recently, we have had the opportunity to observe 1 such case in which bronchoscopy was performed.

11. MacDonald, I. G.: Local and Constitutional Pathology of Bronchial Asthma, Ann. Int. Med. **6**:253-277 (Aug.) 1932.

12. Bubert, H. M., and Warner, C. G.: Fatal Asthma: Report of Case with Bronchial Measurements, J. A. M. A. **104**:1469-1472 (April 27) 1935.

13. Cohen, M. B., and Rudolph, J. A.: Use and Abuse of Drugs in Asthma, J. A. M. A. **98**:1864-1866 (May 28) 1932.

14. Kountz, W. B., and Alexander, H. D.: Death from Bronchial Asthma, Arch. Path. **5**:1003-1019 (June) 1928.

15. Dyspnea Without Obvious Cause, Cabot Case 14161, New England J. Med. **198**:861 (June 7) 1928.

16. Steinberg, B., and Figley, K. D.: Pathology of Asthma, J. Lab. & Clin. Med. **13**:921-942 (July) 1928.

A 49 year old woman was admitted to the medical service on Sept. 5, 1940. There was nothing significant in the past history. She had not been subject to frequent colds.

Six weeks before admission, without any past history of asthma, she began to have nightly attacks of difficulty in breathing, associated with cough. The episodes lasted about one hour. The cough was productive of thick yellow viscid sputum.

The patient was a thin white woman. The blood vessels in the ocular fundi were tortuous. The nasal septum was deviated to the right. The tonsils were present. The blood pressure was 130 systolic and 60 diastolic. The pulse rate was 46, with frequent interpolated extrasystoles. The chest was emphysematous, with audible wheezing; rhonchi were present throughout both sides of the chest. The uterus was fibroid. The patient's temperature on admission was 100.2 F. and her respirations 20.

An electrocardiogram showed a sinal arrhythmia. The urine was normal. Examination of the blood showed the hemoglobin content 93 per cent, the white blood cells 12,900, segmented forms 86, nonsegmented forms 12, lymphocytes 13, monocytes 5 and eosinophils 4. The sputum was negative for tubercle bacilli. A culture of sputum gave a growth of Streptococcus viridans and Micrococcus catarrhalis. The Wassermann reaction of the blood was negative. Skin tests gave positive results for grapefruit and dust.

On admisison the patient was given 10 cc. of theophylline with ethylene diamine U. S. P. intravenously, with relief of dyspnea, considerable clearing of the chest and acceleration of the pulse rate to 100.

During the next ten days she remained in mild status asthmaticus. She was given frequent injections of epinephrine often without relief. When necessary, theophylline with ethylene diamine was given intravenously, with quick but only temporary improvement. The patient received barbiturates and a few doses of codeine (1 grain [0.06 Gm.]) for the alleviation of cough and discomfort. On September 17 she suddenly became much worse. Respiratory difficulty became progressively more marked; epinephrine was given without relief. At 1 p. m. a dose of codeine (1 grain [0.06 Gm.]) was given, without relief. Cyanosis and dyspnea became worse. At 5:45 p. m. she appeared extremely dyspneic and cyanotic. The breath sounds were almost completely absent over the lower lobe of the right lung and very faint on the left. There was no dulness on percussion and no shift of the trachea or heart. The patient was put in an oxygen tent and given theophylline with ethylene diamine intravenously, without avail. She lapsed into semistupor and was rushed to the operating room, where bronchoscopic intervention was performed without anesthesia—none being necessary because of her stupor. At the time, the impression of all who saw here was that she was near death.

As soon as the bronchoscope was passed into the trachea, a large amount of thick tenacious mucopus was aspirated. The tube was then passed into each main bronchus, from which further large amounts of mucopus were aspirated. There was immediate improvement. Ten minutes later the patient was in an oxygen tent, responding well, breathing with scarcely any difficulty, not cyanotic. Breath sounds were coming through fairly well throughout. Improvement continued until, in two hours, the patient was comfortable outside the oxygen tent for short periods. During the next few days she continued to improve, though occasional asthmatic episodes still occurred. There was no recurrence of cyanosis or marked dyspnea.

The bronchoscopic aspiration was life saving.

Effective therapy in status asthmaticus should aim at reduction of secretion and removal of the mucus from the bronchi in order to prevent asphyxia.

The bronchoscope may be a life-saving instrument. In skilful hands it can be passed quickly and without difficulty. For patients who are semistuporous or have already become stuporous, little or no anesthesia is necessary. For those who still have a cough reflex, the use of cocaine with epinephrine or pontocaine with epinephrine or other anesthetic agents does no harm. The shrinkage in the mucous membranes caused by these agents is in itself an aid in opening up the tracheobronchial airway.

CONCLUSIONS

1. The most important factor in causing death in status asthmaticus is obstruction of the tracheobronchial airway by viscid secretion from the mucous glands of the bronchi.

2. This material can be removed by suction through a bronchoscope.

3. Bronchoscopic intervention should be performed as a life-saving measure in cases of status asthmaticus when other measures have proved unsuccessful.

SUMMARY

Eight cases in which death was due to status asthmaticus are presented, with postmortem observations.

The cases reported in the literature in which death was due to status asthmaticus are reviewed.

A plea is made for use of bronchoscopy and suction of obstructing secretions as a life-saving procedure in severe status asthmaticus.

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DERMOID CYSTS OF THE DORSUM OF THE NOSE

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Fistulas and dermoid cysts of the dorsum of the nose are not common. Hagens [1] found that up to 1933 there were only 26 cases reported in the literature. However, this number does not correspond to the actual frequency because small dermoids are not rare among infants and young children. Much more seldom are such tumors to be found in older persons, and even less common are the instances recorded in which the dermoid has produced marked changes of the framework of the nose. In the following cases, the patients are older persons; in 1 of these, marked changes of the bony framework of the nose were observed.

REPORT OF CASES

CASE 1.—L. G. was a 19 year old girl whose birth was described as "non-instrumental." There was no history of deformities in other members of the family, nor were there any deformities or cysts in any part of the body other than the nose. When the patient was 6 months old, her mother noted a swelling just below the bridge of the nose which became soft and felt as if it contained fluid. This slowly grew larger, and in 1933, she visited a physician who thought the swelling was an abscess. He made a midline incision and drained the "abscess." After the operation there was an intermittent seepage of fluid from the center of the wound which never healed.

At the junction of the nasal bones and the triangular cartilage, a small fistula was present from which serum could be expressed. Under local analgesia the linear scar was excised, and the cystic mass was removed from a shallow oval cavity between the nasal bones. The skin was closed with clips, and the following day an isograft of costal cartilage was shaped to fit. The cavity was dusted and the cartilage rolled in sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) powder; the cartilage was placed in the concavity, and the skin was closed by intradermal sutures without drainage.

The pathologic specimen was that of a thin-walled sac filled with several strands of black hair and yellow cheesy material. The sac measured about 1.5 cm. in length, 8 mm. in width and 5 mm. in depth. There was only one opening present, and that was the fistulous tract which extended to the cutaneous surface, measuring 2 mm. in width and 2 cm. in length.

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1. Hagens, E. W.: Congenital Dermoid Cyst and Fistula of the Dorsum of the Nose, Arch. Otolaryng. **28**:399 (Sept.) 1938.

CASE 2.—R. J. was a 19 year old young man whose birth had been normal. Immediately after birth, a nasal deformity as well as a tumor at the glabella was noted. He never noticed a sudden increase or an abnormal appearance of nasal discharge. Both the nasal deformity and the tumor increased as he grew older. His parents and his one brother did not show any abnormalities. The patient was an extremely tall young man, 6 feet and 3 inches (190.5 cm.) in height, and he weighed

Fig. 1.—*A*, *B* and *C*, the patient before operation, *D* and *E*, after operation. The solid lines in *A* indicate the subcutaneous extension of the dermoids; the dotted line indicates the situation of the internal fistula.

195¼ pounds (88.6 Kg.). There was marked deformity of the nose (fig. 1 *A*, *B* and *C*), and on the lower part of the dorsum of the nose there was a fistula the size of a thin pencil. This fistulous opening led toward the root of the nose into a channel which was 2 cm. long and ended blindly. The fistula was entirely lined by skin. Also in the inside of the channel there was skin. The opening of the fistula was surrounded by eczematous skin surrounded by rhagades. The fistula occasionally discharged a clear watery fluid. Toward the tip of the nose, the

fistula continued as a sulcus which extended to the columella. The roof of the fistula consisted of a tumor mass about the size of a plum. It was soft and adherent to the underlying bone. The nares on both sides were normally formed, but there was a little tumor on the mesial margin of the left nostril. There was marked deviation of the septum to the right; the conchae were normal.

Above the glabella, close to the margins of the hairs, there was a tumor covered by skin, similar to that observed on the dorsum of the nose. The tumor was the size of a plum. The epipharynx and the hypopharynx were normal in appearance. Roentgen examination of the skull (fig. 2) revealed well developed sinuses, which were clear. The roofs of the ethmoids were not symmetric, as shown in figure 2. The sella turcica was not enlarged; on the contrary, it was rather small. The findings of the physical examination and of an analysis of the blood and the urine were normal. On Nov. 27, 1940, the midfrontal mass was investigated in another department, first of all by aspiration. However, no fluid

Fig. 2.—Roentgenogram of the patient in case 2. Note the extensive development of the sinuses and the asymmetry of the roofs of the ethmoids.

was obtained. Accordingly, the small mass was excised, and the microscopic examination of the mass revealed a dermoid. Unfortunately, a short time after this operation there was recurrence of the tumor above the glabella.

On Dec. 14, 1940, the nose was operated on under local analgesia (H. B.). A longitudinal incision over the dorsum was made, encircling the fistula at the tip of the nose. The dermoid cyst together with the fistula was removed. The removal was difficult because of firm adhesions between the cyst and its bed. Excessive bleeding also was experienced. It was not possible to remove the cyst in toto since its inner wall extended into a fistula the size of a bean. This fistula led into the nose and was lined by epidermis as a continuation of the dermoid cyst. The dermoid cyst therefore had two openings: one opening externally, close to the tip of the nose, and another leading to the inside of the nose near the junction of the bony and cartilaginous portions of the nose. After the removal of this cyst, another was seen adherent to the left os nasale. Removal of the latter cyst was even more difficult because it not only extended toward the glabella but had also two branches directed toward the mesial part of both supraorbital arches. While

the cyst was being removed from the left os nasale, it ruptured, and sebaceous material exuded.

After removal of both cysts the following anatomic picture was evident: Both alar cartilages were present, but they were not united in the midline. Of the two triangular cartilages, the left was situated almost in the sagittal plane, while the right cartilage was almost entirely absent and replaced by a fistula draining into the nose, close to the anterior part of the quadrangular septal cartilage. The left nasal bone was enormously thickened (it was almost twice the normal thickness) and was situated in the sagittal plane. The right nasal bone was smaller and almost in the sagittal line. Between the two nasal bones, the nasal septum and the connective tissue could be seen. However, at the root of the nose, the two nasal bones were grown together. Between the two nasal bones and the frontal squama there was a marked sulcus.

A piece of subcutaneous tissue was placed into a skin pocket between the mesial crura of the two alar cartilages in order to smooth the sulcus of the tip of the nose. The cavity on the dorsum of the nose was packed with iodoform gauze, and stay sutures were placed. No bony repair was attempted at this time.

Microscopic Examination.—Both tumors were dermoid cysts with a fistula lined by thick ramified epidermis. The walls of both cysts consisted of firm connective tissue and sebaceous glands. In addition, the wall of the upper dermoid cyst contained many muscle fibers which showed cross striation to a great extent.

On December 28, submucous resection of the septum was performed, and an attempt was made to close the fistula of the dermoid cyst which led into the nose. The tumor originating from the left nostril was removed, and its microscopic study revealed many sebaceous glands embedded in firm connective tissue, lined by a normal epidermis.

On Jan. 4, 1941, the dorsum of the nose was reopened under local analgesia. The periosteum was reflected back from both nasal bones. A triangular wedge was removed from the right nasal bone, and both ossa nasalia were thinned. However, on account of the abnormal thickness of the bones, it was not possible to push the nose over to the right. Thus the ossa nasalia were separated from the frontal squama by means of the chisel, and it was then possible to move the nose to the right. In order to maintain the nose in this position, a small piece of isograft was inserted into the defect of the left nasal bone. The periosteum was then sutured back to hold the graft in place. A suture of twenty day chromic catgut was then passed through the right side of the nose externally; another was passed subcutaneously into the left side and pulled over to the right. The catgut was fixed externally on the right side. The upper portion of the wound was closed, and the inferior part was allowed to remain open. Iodoform gauze was inserted, and the nose was pulled to the right by adhesive tape. On January 9, an external nasal mold was fashioned out of dental compound with an opening allowed for drainage from the lower part of nasal incision. The nasal mold was then anchored by strips of adhesive tape. The mold was removed on January 14. On January 23, the dorsum of the nose was reopened. A small remnant of the dermoid over the left nasal bone was excised, and at this time the skin was perforated. Excess tissue was removed bilaterally and reimplanted into the tip of the nose. A small gauze drain was inserted into the skin perforation. The dorsal wound was completely closed with dermal sutures, and a pressure bandage was applied. The patient made an uneventful recovery.

On August 10 there was no recurrence of the dermoid cysts, and the fistula at the tip of the nose was entirely closed (fig. 1 *D* and *E*). There were red scars

on the dorsum of the nose. However, plastic repair of the skin was postponed to permit further observation of the patient for recurrence of the dermoid. There was ample space in both sides of the nose. The fistula into the nose was closed.

COMMENT

To understand the pathologic nature of dermoid cysts of the nasal dorsum it is necessary to recall the anatomic character of this area in embryos as well as in adults. Textbooks of anatomy and embryology furnish this knowledge. However, it is amazing that we could not find a satisfactory description of the anatomy of the dorsum nasi in the newborn, which is of prime importance in the consideration of our subject.

Dorsum Nasi in the Newborn.—According to Gruenwald,[2] the external nose is cartilaginous at birth and forms the anterior wall of the cartilaginous capsule of the nose. Adjacent to the cartilaginous

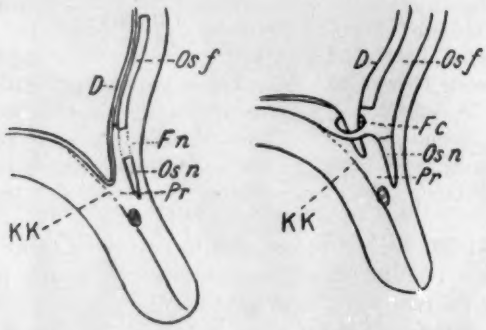

Fig. 3.—Diagram after Gruenwald: *D*, the dura; *Os f*, the frontal bone; *Fn*, fonticulus nasofrontalis; *Os n*, nasal bone; *Pr*, prenasal space; *KK*, cartilaginous capsule of the nose; *Fc*, foramen caecum. The shaded circle indicates the displaced portion of the epidermis.

capsule of the nose, the nasal and the frontal bones develop by intramembranous ossification. Between the paired frontal bones and the nasal bones there is a firm membrane, called by Zuckerkandl the fonticulus nasofrontalis. Behind this fontanel, the anterior wall of the cartilaginous capsule of the nose extends over into the lamina cribrosa. Thus space is formed by the anterior wall of the nasal capsule on the one hand and the nasal bones and the nasofrontal fontanel on the other. This space was called the prenasal space by Gruenwald [2] and is bounded as follows: posteriorly, by the cartilaginous capsule of the nose; anteriorly, by the nasal bones and the nasofrontal fontanel; laterally, by the frontal process of the superior maxilla, and upward, by the dura. In the downward direction, the space passes over into a shallow sulcus which almost extends to the tip of the nose (fig. 3).

2. Gruenwald, L.: Beiträge zur Kenntnis kongenitaler Geschwülste und Missbildungen an Ohr und Nase, Ztschr. f. Ohrenh. **60**:270, 1910.

According to the foregoing description, the prenasal space has no communication with either the nose or the paranasal sinuses, which of course are not developed at this time. Within this space, a teatlike projection of the dura is present. During growth, the nasofrontal fontanel is replaced by the nasal process of the frontal bone, and the teatlike portion of the dura is encircled by a bony canal, the walls of which are made up by the incisura ethmoidalis of the frontal bone. The opening into this canal is termed the foramen caecum. According to Gruenwald,[2] this canal is not situated between the frontal and ethmoid bones; it rather lies within the nasal process of the frontal bone itself.

To illustrate better the anatomy of the nasal dorsum, we present in figure 4 a sagittal section through the external nose of a stillborn male infant which lived fifteen minutes prior to the delivery and died as the result of strangulation of the umbilical cord.

The section demonstrates the cartilaginous lamina cribrosa and the cartilaginous crista galli. The latter is continuous with the anterior wall of the cartilaginous capsule of the nose. On the anterior wall the nasal bone is located. Between the latter and the anterior wall of the cartilaginous capsule of the nose, there is a space which is enlarged in the specimen by an artefact and which contains firm connective tissue serving as an internal periosteum of the nasal bone. This space is termed the prenasal space, and the firm connective tissue is a part of the dura, as can be confirmed by following the strand of connective tissue through the foramen caecum into the dura. Therefore, on the dorsum nasi of newborn infants there is a strand of connective tissue originating from the dura, passing through the foramen caecum and serving as an internal periosteum of the nasal bones. In other words, there are parts of the dura in the dorsum nasi of newborn infants which extend to the inferior margins of the nasal bones.

Pathologic Nature.—According to Ewing,[3] the origin of the dermoids of the scalp is explained

> . . . by an imperfect separation of dura and skin during the intramembranous formation of the cranial bones. As the bone forms between the dura and the skin a portion of the latter may adhere to the dura, become enclosed in bone and form dermoids which lie within the skull and may be connected with the skin by a pedicle.

This explanation clarifies well the dermoids of the nasal dorsum. Figure 4 illustrates the fact that in the area of the nasal dorsum the nasal bones develop as membranous bones between the dural connective tissue and the skin. Consequently, also in the area of the nasal dorsum portions

3. Ewing, J.: Neoplastic Diseases, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

of the skin may adhere to the dura. Stupka [4] expressed the opinion that the dura may pull parts of the ectoderm into the depth. This hypothesis may or may not be correct. Nevertheless, the displaced portion of the

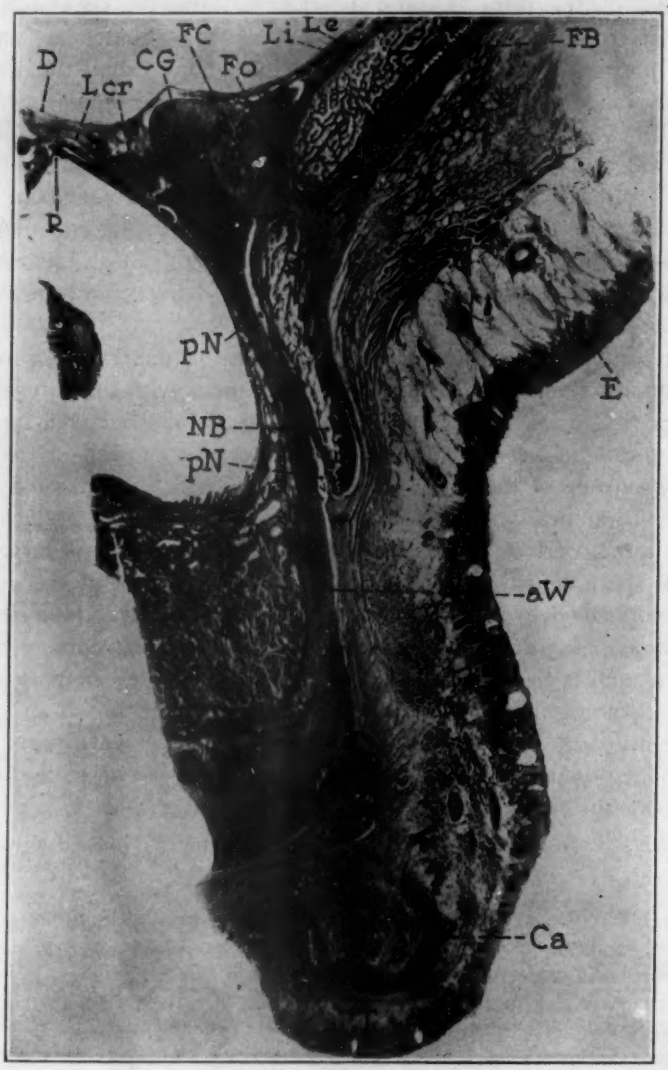

Fig. 4.—Sagittal section through the external nose of a newborn infant. *D*, dura; *R*, roof of the nose; *L cr*, lamina cribrosa; *CG*, crista galli; *FC*, foramen caecum; *Fo*, fonticulus nasofrontalis; *Li*, lamina interna of the dura; *Le*, lamina externa of the dura; *FB*, frontal bone; *E*, epidermis; *aW*, anterior wall of the cartilaginous capsule of the nose; *Ca*, cartilages of the tip of the nose; *T*, tip of the nose; *S*, mucous membrane of the nasal septum; *pN*, prenasal space; *NB*, nasal bone.

4. Stupka, W.: Die Missbildungen der Nase und des Nasenrachenraumes, Berlin, Julius Springer, 1938.

ectoderm is always located within the prenasal space—this for the reason that the membranous bones of the skull develop at the end of the second or the beginning of the third month of the embryonal life, viz., in a period in which the cartilaginous capsule of the nose has already developed. If there is displacement of an ectodermal portion in the area of the nasal dorsum, it can only occur prior to the development of the nasal bones. As a result, the nasal bones develop above the displaced portion of the ectoderm, and thus the dermoid must be situated behind the nasal or frontal bones, viz., in the prenasal space, provided it has any connection at all with these bones. If the displaced portion of the ectoderm retains its connection with the skin, a primary fistula of the dorsum of the nose will result. If this connection disappears, a dermoid cyst develops which in turn may later break through the skin, forming a secondary fistula of the dorsum of the nose.

The fistulas as well as the dermoids have no communication with the nasal cavity or with the cavity of the skull and only exceptionally with the frontal sinus, when they reach an extraordinary size. In our case 1, the patient had such a typical dermoid with a secondary fistula. In this case the dermoid was situated at the inferior part of the nasal bones and did not extend beyond them.

The condition was more complicated in case 2. According to Luongo,[5] dermoid cysts occur at the bridge of the nose along the dorsum nasi (most commonly between the lower margins of the nasal bones and the tip), near the tip of the nose, at the inner angle of the orbit and at the nasofacial sulcus. The patient in case 2 had one dermoid in the middle of the forehead, one at the inner angle of the left orbit (this one extended toward the supraorbital arches on both sides) and a congenital fistula between the lower margins of the nasal bones and the nasal tip. In addition to these deformities there was a wart in the left nostril. A giant-like skeleton combined with a relatively small sella turcica and asymmetry of the roof of the ethmoids were conspicuous.

Only the deformities of the nasal dorsum in case 2 will be discussed. First, the fistula in this case was actually congenital and not secondary (such as was encountered in case 1) because the photographs showing the patient as a baby revealed the presence of the fistula. This congenital fistula had produced diastasis of the alar cartilages, displacement of the left triangular cartilage and probably deviation of the septum. As was pointed out by Bramann,[6] the changes of the nasal framework in these cases are not due to an abnormal development of its anlage but are due

5. Luongo, R. A.: Dermoid Cyst of the Nasal Dorsum, Arch. Otolaryng. **17**: 755 (June) 1933.

6. Bramann, F.: Ueber die Dermoid der Nase, Archiv. f. klin. Chir. **40**:101, 1890.

rather to the pressure of the dermoid. However, the congenital fistula in case 2 did not extend beyond the confines of the nasal bones, as it usually does, but had destroyed the right triangular cartilage, producing a secondary fistula into the nose. It is in this finding that case 2 differs decidedly from the usual findings in similar instances. The explanation for this difference is apparently offered by the large dermoid cyst at the inner angle of the left orbit which did not permit an extension of the congenital fistula toward the glabella.

The dermoid which according to the clinical examination was situated at the inner angle of the orbit had actually developed between the nasal bones within the prenasal space at the root of the nose. It produced diastasis of the nasal bones and marked thickening of the left nasal bone.

Treatment.—It has often been emphasized that cauterization of the fistula is insufficient, inasmuch as Streit [7] found hair follicles deeply embedded in the walls of the fistula (at a depth of 1.5 mm.). It is obvious that under such circumstances the cauterization must be applied rather extensively to avoid an early recurrence. Consequently, excision of the fistula or the cyst is the method of choice. Excision is performed with a cold scalpel or by means of surgical diathermy; at any rate, it must be performed thoroughly. We employed the scalpel in order to avoid injury to cartilage and bone. Many rhinologists are reluctant to perforate the mucous membrane of the nose during this type of operation. There is no doubt that such an occurrence is best avoided; however, there is no great danger if it does occur. The patient in case 2 had a large fistula into the nose, inasmuch as the dermoid had destroyed the entire right triangular cartilage. Nevertheless, this large fistula closed spontaneously after the operation.

The result of operation as far as cosmesis is concerned was excellent in case 1 but not so good in case 2 because the scars at the dorsum of the nose were prominent, probably on account of the previous infections. However, plastic repair of the skin as well as that of the framework of the nose had to be postponed until sufficient time had elapsed to be certain that no recurrence of the dermoid would occur. These patients were observed to August 1941, and no recurrences were noticed.

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7. Streit, H.: Beitrag zur medianen Nasenfistel, Arch. f. Laryng. u. Rhin. **24**:454, 1911.

VESTIBULAR KINETOVISUAL FUNCTION AND KINETIC VISION

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In this article I shall describe two functions regarding which I believe I have obtained new information: First, the kinetovisual function of the vestibular apparatus and, second, kinetic vision. Both of these functions come into action during vision of moving objects.

The kinetovisual function of the vestibular apparatus cooperates with the visual apparatus in vision of moving objects during movements of the head. I believe this function to have been hitherto unknown, as I do not find it described in any textbook of physiology, although its existence has been suspected by almost every investigator. The reflexes which govern this function, such as the compensatory movements of the eyes, the *Gegenrollung,* or counter rotation, and the perrotatory nystagmus, have been thoroughly studied, and the purpose of these reflexes, to retain the orientation of the visual field on the retina, is also known. I shall show a relationship between these reflexes which serves as the basis for a function of the vestibular apparatus hitherto not described —that is, one to permit vision during movement of the head. This relationship has never been demonstrated before.

Kinetic vision is designed to permit vision of objects actually moving or seeming to move in front of the eyes. This function cooperates with the kinetovisual function of the vestibular apparatus when the displacement of external objects from in front of the eyes is caused by movement of the head and cooperates with the retinal reflexes when the external objects move of their own accord.

I shall begin by describing the mechanism of the visual function; then I shall consider the peculiarities of the vision of still and moving objects and the different mechanisms which assist in the vision of moving objects, and, last, I shall describe the two functions mentioned in the first paragraph.

THE MECHANISM OF VISION

The mechanism of vision is rather complicated, and to facilitate its description I shall refer only to those particulars about which it is necessary to know in order to understand how vision is accomplished.

When there is vision, the rays of light reflected by the external objects are directed through the refractive surfaces of the eye to the

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retina, forming there the optical image of the external objects. The effect of the waves of light falling on the retina is to set up a series of nerve impulses in the fibers of the optic nerve which are transmitted to the visual center of the cortex, where the visual sensation is perceived.

The optical image must act on the retina during a minimum period of one twentieth of a second in order to produce a stimulation capable of giving rise to a normal visual sensation. It must also remain fixed on the retina while the stimulation takes place, for if it slides over the surface of the retina during this time, the visual sensation produced will be blurred, giving the impression of motion.

This description corresponds approximately to that which is found in almost every textbook of physiology. It may be observed that no distinction is made between the vision of still and that of moving objects, from which one might conclude that both kinds of vision are accomplished in the same manner. But as soon as this mechanism is analyzed, even superficially, one appreciates that the vision of moving objects has not heretofore been thoroughly studied.

This description explains very well the vision that occurs when the head and the external object are motionless. In these cases the optical image remains stationary on the retina, and the stimulation is carried on in a normal manner, as the conditions required to produce it are present, giving rise to a normal visual perception.

But it does not explain satisfactorily that vision of objects which occurs during rotation or inclination of the head, during transportation of the body or during movement of external objects. In these cases one might suppose that the optical image would slide on the retina, so that stimulation would be deficient, as the optical image would not remain fixed on the retina the required length of time to make an impression, and consequently that the visual perception would be blurred or confused. But this is not what really happens, as the vision of external objects in the cases in question occurs perfectly well, notwithstanding the supposed sliding of the optical images on the retinas. This disagreement between theory and fact leads one to believe that the visual apparatus must bring into action some auxiliary mechanism to immobilize the optical images on the retinas, because otherwise the vision of objects moving or seeming to move from in front of the eyes could not be explained according to mechanical laws.

These considerations led me to investigate this mechanism. I took into consideration each of the causes producing real or apparent movement of external objects from in front of the sight, then analyzed how vision of those objects might be accomplished in each case.

IMMOBILIZATION DURING ROTATION OF THE HEAD

When vision is accomplished during a rotatory movement of the head, that is, during a movement around any axis situated at the level of the neck, two ocular manifestations are observed which do not occur when vision takes place while the head is motionless. These are movement of the panorama and perrotatory nystagmus.

The first ocular manifestation is the one observed when the head is turned around any axis—that is, the panorama, or external objects, are seen as though moving in front of the sight, to which visual impression I have given the name "movement of the panorama."

In order to study this manifestation, the observer may subject himself to rotations in a revolving chair around different axes and in different directions and may observe how the panorama moves in each case. When this is done, it is observed that the panorama produces the impression of a gigantic sphere that turns around one. It is easy to describe the details of the sphere's movement by stating the position of its axis or plane of rotation and the direction of its movement.

When the head is turned on a horizontal plane, the panorama appears to turn horizontally in the opposite direction; when the head is turned on a vertical plane, the panorama is seen to move vertically in the opposite direction, and when the head is turned around any other plane of rotation, the panorama will always appear to turn around the same plane of rotation but in an opposite direction. Therefore, it may be said that during rotatory movements of the head the panorama appears to turn around in the same plane but in the opposite direction to that of the rotation of the head. One may express the same idea, referring it to the axis of rotation, by saying that the panorama turns around a parallel axis and in a direction opposite to that of the rotation of the head.

The other ocular manifestation appearing during rotatory movements of the head is perrotatory nystagmus, which is a rhythmic movement of the eyes, composed of two partial rotatory movements around the same axis and in opposite directions, which occur continuously while the nystagmus lasts.

It is produced by the stimulation of the semicircular apparatus. Whenever the head undergoes a rotatory movement, the endolymph is shifted within the semicircular canals, thus stimulating the ampullar endings of the vestibular nerve. This stimulation gives rise to several reflexes, one of which is the nystagmus.

This reflex appears every time the head rotates, even if the rotation is slow or of short amplitude. It may be demonstrated by turning a person on a rotatory chair and also by carrying out the following experiment: Place the finger tips lightly over the closed eyelids, so as to be able to appreciate by touch any displacement of the eye. As soon as

the head is turned in any direction, one feels under the finger tips the beatings of the perrotatory nystagmus produced by the rotation of the head.

Sometimes the perrotatory nystagmus does not appear during a rotatory movement of the head. This happens when the person fixes his gaze on any external object during the turning of the head, for this action prevents or inhibits the nystagmus. This affirmation may be proved by the following experiment, which demonstrates the inhibitory effect of fixation of the sight on the nystagmus: A person is submitted to rotation and ordered to fix his gaze on one of his fingers, placed about 2 feet (61 cm.) in front of his eyes; it is then observed that the nystagmus is absent notwithstanding a rapid rotation and that it reappears as soon as the fixation of the gaze is stopped. For this reason Bartel's lenses of 20 diopters should be employed whenever one investigates nystagmus, as this procedure prevents fixation of the gaze.

The position of the axis of rotation and the direction of the nystagmus are related to the similar characteristics of the rotatory movements of the head which produce the nystagmus. These relations may be determined by subjecting a person to rotation about different axes and in different directions and observing the results obtained in each case.

When the head is turned horizontally, the nystagmus will be horizontal; when it is turned vertically, the nystagmus will be vertical, and when it is turned in any other plane, the nystagmus will be of a similar plane. Accordingly, one may say that the nystagmus turns around the same axis or plane as that of the rotatory movement of the head. If one repeats these experiments in order to observe the direction of each of the components of the nystagmus, one appreciates that in every case the slow component of the nystagmus is directed in an opposite and the rapid component in the same direction as that of the rotatior of the head. One may summarize the characteristics of perrotatory nystagmus by saying that the nystagmus turns around the axis of rotation of the head, with its slow component in an opposite direction and the rapid component in the same direction. These relations between the characteristics of the nystagmus and those of the rotation of the head were determined by me and were reported in another paper.[1]

Thus nystagmus forces the eyeballs to move with the same character of motion as the head whenever the latter undergoes a rotatory movement: The eyeballs will be moved around the axis of rotation of the head, with a slow movement in the opposite direction and with a rapid movement in the same direction as that of the rotation of the head.

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1. Arellano, E. R.: Simplification of the Rules Concerning Planes and Directional Sense of Any Nystagmus Consequent upon Rotation of the Head, Together with Explanation of Purpose of Nystagmus, Acta oto-laryng. **26**:367, 1938.

When one compares the two ocular manifestations appearing during rotatory movements of the head, some interesting facts are observed:

1. The slow movement and the movement of the panorama before one's sight occur around axes parallel to that of the head's rotation and in a direction opposite to that of the movement of the head.

2. The slow movement of the eye is periodic, being interrupted each time that a new rapid movement appears. But the movement of the panorama is continuous, that is, both manifestations move in a similar manner only during the slow movement of the eyes.

3. The rapid movement and the movement of the panorama occur around axes parallel to that of the rotation of the head but in opposite directions.

4. The rapid movement of the eye is periodic, while that of the panorama is continuous, so that each time the eye moves in a direction opposite to that of the panorama it is directed to a new sector of the panorama, that is, to the one placed each time in front of the eyes.

As the nystagmic movements of the eyes alternate while the head moves, every time a slow movement of the eye occurs the eye follows the sector of the panorama placed in front of it and every time a rapid movement of the eye occurs the eye is directed toward the next sector of the panorama which happens to be placed in front of it as a consequence of the continuity of the movement of the panorama.

This comparison between the two ocular manifestations shows that the nystagmic movements of the eyes caused by the rotation of the head oblige the eyes to follow the movement of the different sectors of the panorama as they cross in front of the sight.

Now that one knows the two ocular manifestations produced by a rotatory movement of the head and the relations between them, one may understand the mechanism of vision during this kind of movement.

Each time the eye follows a sector of the panorama, impelled by the slow component, the eye and the panorama are displaced in the same direction, and the optical image of that sector of the panorama remains stationary on the retina, and thus the retina may be stimulated in such a manner as to produce a normal visual perception. Each time the eye is directed to the next sector of the panorama by virtue of the rapid component, the optical image will move very rapidly on the retina, with a speed equal to the sum of the velocities of the two manifestations, owing to the fact that the eyes and the panorama move in opposite directions. On account of the swiftness of the displacement of the optical image, stimulation will not occur and no visual perception will be produced.

In this manner, the retinas are stimulated only during the slow movements of the eyes by the optical images of the different sectors

of the panorama that cross in front of the eyes. The rapid movements of the eyes do not intervene in the stimulation of the retinas, being intended to bring the eyes to the normal position each time they are abducted from this position, the rapid component thus setting them ready for the action of the next slow component.

The retinas thus are stimulated by stationary optical images of the different sectors of the moving panorama. If one could register on a photographic film the different impressions received by each retina, one would get the same kind of pictures one observes in a cinematographic film, on which are registered different "still" pictures of a moving panorama or scene.

Once the optical image is immobilized on the retina and the stimulation carried on, then vision is accomplished in the same manner as with still objects; that is, the retinal impressions are transmitted to the cortical centers where they are normally perceived. As the perception of each of the optical images lasts a certain length of time, the images fuse with one another, giving the impression of a continuous sight.

I have just explained how vision during the movements of the head is made possible by the assistance of perrotatory nystagmus, without which this kind of vision could not be accomplished. This hypothesis may be demonstrated experimentally.

If it is true that perrotatory nystagmus is the mechanism which permits vision during rotation of the head, then one may suppose that vision cannot take place during movement of the head if nystagmus is absent. In order to find out if this supposition corresponds to fact, one may order a person to fix his gaze on one of his fingers, placed about 2 feet (61 cm.) from his eyes, in order to prevent perrotatory nystagmus, and then to turn his head from one side to the other. An observer in front of the subject will note that perrotatory nystagmus is absent while the subject turns his head, and the subject will see a blurred or confused panorama turning in front of him. If one repeats the experiment without obtaining fixation of the gaze, perrotatory nystagmus will be observed during the turning of the head and the subject will see clearly the panorama turning in front of him. This experiment shows that when nystagmus is absent vision is imperfect during rotation of the head and that when nystagmus is present vision is accomplished normally or, in other words, that during movement of the head vision may be accomplished only through intervention of perrotatory nystagmus.

IMMOBILIZATION DURING INCLINATION OF THE HEAD

One must investigate also how the optical image is immobilized on the retina when vision takes place during inclinatory movement of the head. Inclination of the head is understood to mean movement

which inclines the vertical axis of the head in any direction, as when the head changes its position or, while one is walking, when the vertical axis is inclined in different directions, always returning to the normal position of equilibrium.

When vision takes place during an inclinatory movement of the head, two ocular manifestations are observed which do not appear when the head remains motionless. These are: movement of the panorama and compensatory movement of the eyes.

Movement of the panorama always occurs when the vertical axis of the head is inclined. The characteristics of this manifestation are determined by observing the manner in which the panorama moves in front of the sight when the head is inclined in several directions. One finds that the panorama always moves very slowly for a short distance in a direction opposite to that of the inclination of the head.

The other ocular manifestation that appears during the inclination of the head is the compensatory movement of the eyes, called the otolithic reflex, which is made up of slow rotatory movements of the eyes of short amplitude around one of the axes perpendicular to the vertical axis of the head—that is, around the sagittal or the frontal axis. The compensatory movement around the sagittal axis (frontal plane) is observed when the vertical axis of the head is inclined to the right or the left. This kind of ocular movement has been known for a long time and was studied by Bárány,[2] being called *Gegenrollung,* or counter-rotation. As this is difficult to observe, one should take note of some characteristic mark of the iris and then incline the head slowly in any direction. As soon as the compensatory movement appears, the iris mark will be seen to change its position slightly in relation to the angle of the eyelids.

Compensatory movement around the transverse axis of the head (vertical plane) is observed when the head is inclined forward or backward. This compensatory movement, unlike nystagmus, is little known, and although it has been studied by several investigators (Quix,[3a] Magnus [3b] and others), it has not yet received a specific name. To observe it, one inclines the head of the subject forward or backward. The eyes will be seen to move very slowly upward or downward.

The compensatory movements of the eyes are produced by the stimulation of the otolithic apparatus. It is supposed that when the vertical axis of the head is inclined, gravitation produces a displacement of the otoliths which modifies the pressure or the tension exerted by

2. Bárány, R.: Physiologie und Pathologie (Funktions-Prüfung) des Bogengangapparates beim Menschen, Leipzig, Franz Deuticke, 1907.

3. (*a*) Quix, F. H.: Les méthodes d'examen de l'organe vestibulaire, Arch. internat. de laryng. **35**:133, 1929. (*b*) Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924.

the otoliths on their respective maculas, thus producing stimulation of the macular endings of the vestibular nerve in this apparatus.

To determine the characteristics of these compensatory movements one should examine the subject with Bartel's lenses, in order to avoid the ocular movements produced by fixation of the gaze, and then the head should be inclined toward the sides and forward and backward while one observes the peculiarities of these movements. In this manner it will be observed that the two compensatory movements of the eyes occur in the same manner—that is, the eye moves very slowly around the axis of the inclination of the head and in an opposite direction.

On comparing the two ocular manifestations occurring during the inclination of the head, one observes that they possess the same characteristics; that is, the eye turns slowly around the axis and in the direction opposite to that of the inclination of the head. This similarity of characteristics shows clearly that whenever the otolithic apparatus is stimulated by an inclination of the head the compensatory movements of the eyes produced by this stimulation will make the eyes move exactly in the same manner as the panorama in front of the eyes. Thus the optical image of the panorama which would slide over the retina if the eye were stationary during the inclination of the head now will remain fixed on the retina because the eye and the panorama are moving in a similar manner. Once the optical image is immobilized on the retina by this procedure, the impression of the retina will be transmitted to the brain in the same manner as during vision of still objects.

IMMOBILIZATION OF OPTICAL IMAGES DURING TRANSPORTATION OF THE BODY AND DURING MOVING OF THE OBJECTS OF VISION

Two other causes, besides movements of the head, produce sliding of the optical image on the retina; these are the transportation of the body, as in a car or a train, and the movements of external objects, such as those of a running horse, a cascade, a flying bird and the hand. I shall now consider how the optical image is held stationary on the retina when vision takes place during these eventualities.

When the objects move in front of the eyes, the head remaining motionless, the vestibular apparatus is not stimulated, so that the reflexes from the semicircular canal and the otoliths previously described are not produced. As the vestibular reflexes designed to immobilize the optical images on the retinas do not come into action, one supposes that the image slides over the retina and produces an imperfect visual sensation. But this is not what happens, as vision is carried on perfectly in these circumstances. One must suppose that there exists another mechanism, which immobilizes the moving optical images when the head remains stationary. This is the point I shall now explain.

If a person while walking fixes his gaze on any object, it is observed that his eyes follow the displacement of that external object in relation to his sight without conscious effort. The same thing happens if he fixes his gaze on an object being moved from one side to the other in front of his eyes, as the displacement of the object is followed by the eyes. These observations indicate that the fixation of the gaze seems to force the eyes to follow the movement of the object in front of them.

It is also observed that when the eyes cannot continue following the displacement of the panorama because of the limitations of the ocular movements inside the orbit, the person unconsciously turns his head in the direction in which the object escapes from the sight, helping in this manner the fixation of the gaze.

These considerations lead one to suppose that fixation of the gaze and movements of the head could be the mechanisms employed by vision to preserve the image of the visual field on the retina in those cases in which the panorama moves in front of the eyes while the head is at rest and consequently without the assistance of vestibular reflexes.

There are also other reasons which lead one to think in this manner. Experiments made by many investigators, in which lower animals were submitted to different kinds of movements, have shown that there are certain reactions of the head, eyes and body which tend to preserve the orientation of the visual field on the retinas during these movements. Most of these reactions are produced by the stimulation of the vestibular apparatus caused by the movements to which the animal is submitted, as may be demonstrated by the fact that they disappear when the vestibular apparatus is destroyed. But at the same time other reactions are also observed which serve the same purpose, such as the cephalic and the optokinetic nystagmus which are produced by stimulation of the retina. These reflexes may be demonstrated by turning a labyrinthless animal, such as a reptile, in which they appear during rotation, or by displacing an artificial panorama in front of the eyes while the animal is in repose. The reflexes disappear as soon as the eyes of the animal are covered (Maxwell [4]).

These experiments seem to show that in lower animals the reflexes intended to conserve the visual fields on the retinas generally proceed from vestibular sources but that they also replace other reflexes of retinal origin intended to fulfil the same purpose in cases in which the vestibular apparatus is not stimulated.

I suppose the same thing occurs in human beings—that when the head is held motionless and the panorama moves in front of the sight, the vestibular apparatus is not stimulated and then the retinal reflexes,

4. Maxwell, S. S.: Labyrinth and Equilibrium, Philadelphia, J. B. Lippincott Company, 1923.

fixation of the sight and cephalic movements, come into play in order to obtain the same result, that is, to hold the visual field stationary on the retinas.

This supposition is also supported by the fact that when the sight is left errant (*vista vaga*), that is, when there is no fixation of the gaze, the moving panorama will be seen blurred or confused, giving an impression of movement, which fact demonstrates the real value of fixation of the gaze.

The real nature of this reflex is not clearly known, but from what I have been able to find out, it can be explained in the following manner: It is supposed that when the optical image of an object falls on the periphery of the retina, where it is not clearly seen, it gives rise to a retinal reflex which directs the eyes toward the object in such a manner as to bring the optical image to the fovea centralis, where it is clearly seen. This reflex is not produced while the image remains centered on the fovea. This is the reflex which keeps the eyes continually in motion, so as to bring each object, as one pays special attention to it, into the central field of vision.

According to these ideas, one should suppose that when an object focused on the fovea begins to move and its optical image is displaced toward the periphery of the field of vision it will give rise to the retinal reflex which directs the eyes toward the moving object. This reflex will be produced continuously while the image acts on the peripheral part of the retina, so that the eyes will automatically follow the movement of the object while it lasts.

As soon as the moving panorama is followed by the eyes, forced by the action of the retinal reflexes, the optical image will remain stationary on the retina; hence, vision is accomplished in the same manner as with still objects.

KINETIC VISION

At the beginning of this article I stated that the descriptions of the mechanics of vision in the textbooks of physiology did not explain satisfactorily the vision of moving objects, but now that I have made a study of this subject I believe I may offer a more reasonable explanation of this kind of vision.

I have given the name "kinetic vision" to the vision of objects that appear to move or do move. The movement, apparent or actual, of external objects is produced by several causes, such as rotation or inclination of the head, transfer displacements of the organism and movements of the external objects. Whenever an object appears to move or does move in front of the sight, its optical image slides over the surface of the retina, causing deficient stimulation and consequently confused or blurred visual perception.

The moving optical image must be immobilized on the retina in order that it may effect normal stimulation and produce clear visual perception. This immobilization of the optical image is carried out by several mechanisms according to the cause of the sliding of the optical image.

When the movement of the optical image is produced by rotation of the head, the panorama turns around the axis of rotation of the head but in the opposite direction. The stimulation of the semicircular canal apparatus produced by the rotation of the head gives rise to nystagmus, which communicates two different rotatory movements to the eyes around the axis of rotation of the head. One of these moves the eyes slowly in the same direction as the panoramic sector which crosses in front of the eyes; the other forces the eyes to return rapidly to the original position, in order to allow the next slow movement of the eyes to follow the next panoramic sector appearing in front of the eyes. In this manner, as the eyes follow the different panoramic sectors which cross in front of them, their respective optical images will be held stationary on the retinas the required length of time to produce satisfactory retinal stimulation.

When sliding of the optical image on the retina is produced by inclination of the head, the panorama moves slowly in the direction opposite to that of the inclination of the vertical axis. The otolithic apparatus, on being stimulated by the inclination of the head, gives rise to compensatory movements of the eyes. The eyes move in the same manner as the panorama, that is, slowly, in an opposite direction to that of the inclination of the head. As the eye and the panorama move in the same manner, the optical image will remain stationary on the retina while the retinal stimulation is accomplished.

When the sliding of the optical image is produced, not by a movement of the head but by a transfer movement of the organism or by a movement of the external object, the panorama moves also in front of the eyes without following any given rule. This movement of the panorama may not be followed by the ocular movements produced by the vestibular reflexes, as the eye is not stimulated by movement of the head. While the sight is not fixed on any particular object, the eye remains stationary in the orbital cavity and the optical image of the moving objects slides on the retina, giving rise to a blurred visual perception. But as soon as fixation of the gaze and cephalic movements come into action, the eyes are forced to follow the moving panorama or object, and in this manner the optical images will be held stationary on the retinas while the stimulation takes place.

Once the immobilization of the optical images is accomplished by any of the mechanisms already explained, the stimulation of the retinas takes place normally and vision continues in the same manner as with still objects.

The other function described in this article is the kinetovisual function of the vestibular apparatus, which cooperates with the visual apparatus in vision occurring during movements of the head. I believe that this cooperation in kinetic vision deserves to be considered as another function of the vestibular apparatus, in the same manner that the cooperation of this apparatus with other organs in the maintenance of the equilibrium of the body is considered as a function of this organ.

The kinetovisual function immobilizes the optical images that otherwise would be displaced by movements of the head. This result is obtained when the eyes move in the same manner as the panorama moves, impelled by the vestibular reflexes caused by the movements of the head, that is, by the reflexes produced from the semicircular canals by the rotation of the head and the reflexes produced from the otoliths by its inclinations.

The action of the vestibular apparatus in kinetic vision may be compared to that of an eye stabilizer, serving to keep the eyes in the same position or orientation with relation to the panorama during the movements of the head, in order to maintain the optical images, centered on the retinas while the stimulation is affected. I shall not enter into the description of this function, as I described it when summarizing the factors in kinetic vision.

On comparing kinetic vision or vision of moving objects with cinematography or photography of moving objects one observes many interesting facts.

Both require the obtaining of a series of successive still pictures of a moving object or panorama, in order to produce the illusion of continuous vision.

The moving picture camera gets still pictures of a moving object or panorama, based on the speed of the sensitive film. This procedure stops the movement of the optical image focused on the film. Even the image of a flying bullet can be immobilized by using very sensitive films. The visual apparatus is not able to use this procedure, as the speed of the retina is not more than one twentieth of a second, not sufficient to stop the motion of the optical image reflected on the retina. This fact is known to all photographers, as they cannot take a picture of a moving object with a camera having a shutter of one twentieth of a second. But the visual apparatus may immobilize the optical images on the retinas by moving the eyes as the external objects move in front of the eyes, thus holding the optical images stationary on the retinas. This is the procedure imployed in sky photography, in which the movement of the stars is followed with the telescope by means of a clockwork mechanism, and thus the optical image of the stars is immobilized on the photographic film while the impression requiring a long exposure is

made. The ocular movements producing the immobilization are caused by different reflexes. When the displacement of the optical image of external objects is caused by a movement of the head, the vestibular reflexes come into action, moving the eyes in the same manner as the panorama in front of the eyes appears to move. When the movement of the optical image of external objects is due to any cause other than a movement of the head, the retinal reflexes move the eyes in the same manner as the objects move.

The successive views required to produce the illusion of continuous motion are obtained by a different mechanism in each procedure. In cinematography this is achieved by very rapidly passing the film behind the lens, in order to get many pictures of the moving panorama. While the film is being passed, a revolving shutter closes the lens each time a new frame is substituted, in order to prevent the sliding of the optical image on the film while the change is being made. Otherwise, the film would register movement and a blurred picture would be produced. In kinetic vision no special apparatus is required to change the film, as the sensitivity of the film is restored very rapidly after each impression. The revolving shutter, which prevents the impression of the sensitive film by a moving optical image, is not required in kinetic vision, as the sliding of the image on the retina which occurs during rapid movements of the eyes will not cause any impression on the retinas, because the sensitivity of this structure is low. Thus, it is seen that in those cases in which it is necessary to prevent the consequences of movement of the optical image on the retina rapid movements of the eyes are substituted for the action of the revolving shutter.

I believe that the end result in kinetic vision is based on the same mechanical principles that are used in cinematography—that is, one obtains the vision of moving objects by means of a series of still pictures.

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SYNDROME OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY

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In order to evaluate the full significance of the syndrome of the posterior inferior cerebellar artery, a comprehensive knowledge of the anatomy of the region involved is of paramount importance.

The vertebral artery arises from the first branch of the subclavian artery on each side. The course of the artery may be divided into four portions. The fourth portion extends from its entrance into the vertebral canal, through the foramen magnum, to the point where it unites with its fellow of the opposite side to form the basilar artery (fig. 1). During its course about the spinal cord the artery enters and traverses the arachnoid and, gaining the subarachnoid space, passes gradually upward and inward to the middle of the caudal border of the ventral surface of the pons, where it meets and anastomoses with its fellow.

The medulla oblongata is supplied by the posterior inferior cerebellar and by the anterior spinal artery, and sometimes by branches from the basilar artery. Slightly above the posterior inferior cerebellar artery is the anterior spinal branch of the vertebral artery. This vessel descends along the anterior median fissure of the medulla oblongata and sends branches into the posterior longitudinal bundles, the nuclei of the twelfth nerve and other nerves and the pyramid. When these are occluded, symptoms referable to the central and anterior parts of the medulla oblongata will be present. This gives rise to a number of interesting syndromes, which, because of their importance, will be briefly discussed.

The posterior inferior cerebellar artery is the longest branch of the vertebral artery. It arises opposite the olive before the parent vessel unites with its fellow. Winding backward around the olive, it ascends behind the roots of the glossopharyngeal and vagus nerves to the vallecula of the cerebellum, where it divides into a medial and a lateral branch, which supply the cerebellar lobes. These branches anastomose freely with the anterior inferior cerebellar and superior cerebellar branches of the basilar artery. The main trunk of the artery, however, supplies other branches, which are end arteries to the medulla and the

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dentate nucleus of the cerebellum. The latter branches, because they are end arteries, are of fundamental importance in the production of the syndrome.

Occlusion of the posterior inferior cerebellar artery completely shuts off the blood supply to the structures in the dorsolateral aspect of the medulla but only incompletely interferes with the blood supply to the cerebellum, with the exception of the dentate nucleus, for its branches

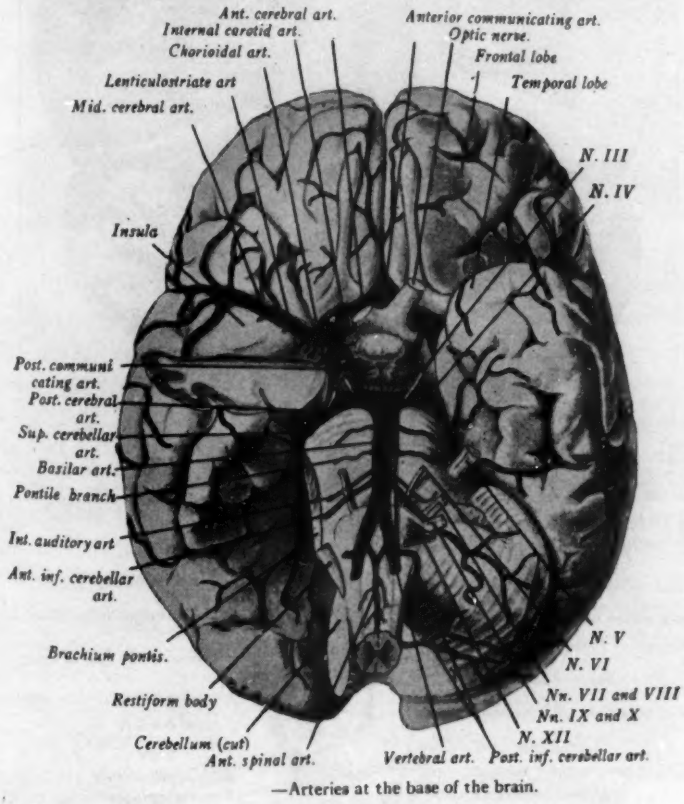

Fig. 1.—Arteries at the base of the brain.

to the cerebellum are not end arteries (Spillane [1]). The probable cause of occlusion of the artery is a thrombus or an embolus. The predisposing cause may be cerebral arteriosclerosis, neurosyphilis or an infectious process or changes in the circulating medium (Riley [2]).

The syndrome of the posterior inferior cerebellar artery is not common, but is by no means rare. The neurologic division of Bellevue

1. Spillane, J. D.: Posterior Superior Cerebellar Artery Thrombosis, Bull. Neurol. Inst. New York **6**:529, 1937.

2. Riley, H. A.: Personal communication to the author.

Hospital reports as many as 12 cases a year (Wortis [3]). Figure 2 shows the gross appearance of the medulla oblongata in cross section, indicating the area destroyed by the complete occlusion of the posterior

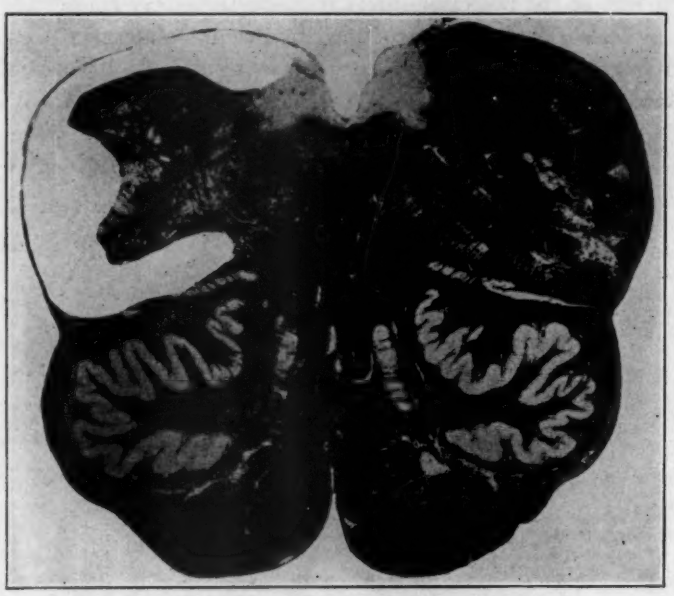

Fig. 2.—Gross appearance of the medulla oblongata in cross section, showing the area destroyed by the complete occlusion of the posterior inferior cerebellar artery (Tilney and Riley [4]).

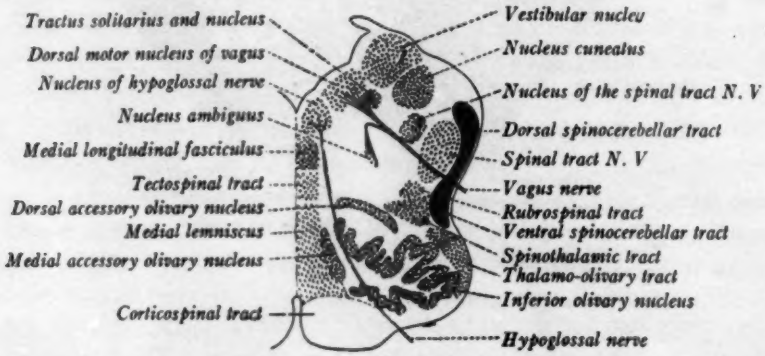

Fig. 3.—Diagram showing location of the nuclei and fiber tracts of the medulla oblongata at the level of the olive.

inferior cerebellar artery (Tilney and Riley [4]). Figure 3 is a diagram of a cross section of the medulla oblongata at the level of the olive,

3. Wortis, B. S.: Personal communication to the author.

4. Tilney, F., and Riley, H. A.: The Form and Functions of the Central Nervous System, New York, Paul B. Hoeber, 1923.

showing the location of the nuclei and fiber tracts. The tracts and nuclei involved in the occlusion of the posterior inferior cerebellar artery are the ventral spinocerebellar tract (Flechsig), the dorsal spinocerebellar tract, the spinal tract of the trigeminal nerve, the lateral spinothalamic tract, the nucleus ambiguus and a group of cells dorsal to the nucleus ambiguus, recognized by some neuroanatomists as the pupillary center. The vestibular nucleus is also involved; the nucleus triangularis is in this area, but Deiters' nucleus is situated a little higher. It is important to note that the nucleus cuneatus, the nucleus of the hypoglossal nerve, the medial longitudinal fasciculus, the medial lemniscus and the accessory olivary nuclei are not involved in the syndrome.

SYNDROME OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY

The involvement of the dorsal and ventral spinocerebellar tracts accounts for the ipsilateral lateropulsion, hemiasynergia and hemiataxia. The patient has a feeling of falling to the side of the lesion. He past points in the direction of the lesion, presents adiadokokinesis and tremor and cannot coordinate the movements of the upper and lower extremities.

The dorsal spinocerebellar tract comes from the spinal cord and lies along the periphery of the medulla oblongata near the posterior lateral sulcus. At the level of the lower part of the olive it inclines dorsally, passing over the surface of the spinal tract of the trigeminal nerve to reach the restiform body. Between this tract and the olive the ventral spinocerebellar tract can be traced in a superficial position.

The ipsilateral facial anesthesia, including all types of sensibility, is due to the involvement of the spinal tract of the trigeminal nerve. This tract is formed by the descending branches of the sensory fibers of the trigeminal nerve. The tract lies along the lateral side of its nucleus and is superficial except so far as it is covered by the external arcuate fibers, the dorsal spinocerebellar tract, and the restiform body.

In most cases the syndrome of the posterior inferior cerebellar artery is ushered in with vertigo. This is due to the sudden irritation of one of the vestibular nuclei, namely, the triangularis. The dizziness soon subsides because there is a compensatory reaction from the central nucleus on the opposite side. The central nystagmus, which is present, is constant as long as the irritation of the nucleus is present and in due time is not associated with dizziness. The slow component of the nystagmus is directed toward the lesion. Caloric irrigation of the ear on the side of the lesion will always produce normal peripheral nystagmus, with its normal concomitant dizziness and past pointing.

The contralateral loss of pain and temperature sensation in the arm, trunk, leg and neck is due to the involvement of the lateral spinothalamic tract, which conveys impulses for pain and temperature from the opposite

side of the body. From cells in the posterior column fibers arise which cross to the opposite side of the cord in the anterior white commissure and ascend in the lateral spinothalamic tract to end in the thalamus.

The ipsilateral paralysis of the soft palate and vocal cord is due to the involvement of the nucleus ambiguus on the side of the lesion. This nucleus is a long column of nerve cells which give origin to the motor fibers that run by way of the glossopharyngeal, vagus and accessory nerves to supply the musculature of the pharynx and larynx. The nucleus is located in the reticular formation of both the open and the closed portion of the medulla. Dysphagia in this syndrome is due to involvement of the nucleus ambiguus. The pharyngeal muscles are supplied by the pharyngeal plexus, which is derived from the glossopharyngeal, the vagus and the spinal accessory nerve. A complete unilateral lesion of the vagus nerve may in itself lead to severe and persistent dysphagia.

The rubrospinal tract (tract of Monakow) serves for the transmission of impulses which have to do with synergic and associated automatic control. It is situated near the center of the mesencephalon, crosses the median plane and descends into the spinal cord, within which some of the fibers can be traced to the sacral region. Their collateral and terminal branches end within the anterior column in relation to the primary motor neurons.

The spinotectal tract consists of fibers which arise from the cells in the posterior gray column and which, after crossing, ascend in the lateral funiculus, in company with those of the lateral spinothalamic path, to end in the roof of the mesencephalon (Ranson [5]). The function of this tract is unknown.

Horner's syndrome, i. e., drooping of the upper lid, narrowing of the palpebral fissure and miosis, is often associated with this syndrome. The pupillary center is considered by most neuroanatomists to consist of a group of cells situated dorsal to the nucleus ambiguus.

The nucleus of the tractus solitarius is the nucleus of reception of the sensory fibers of the facial, glossopharyngeal and vagus nerves. The tractus solitarius can be traced through almost the entire length of the medulla. The central receiving station for the sense of taste is the nucleus of the tractus solitarius. The glossopharyngeal fibers contribute chiefly to this nucleus.

The dorsal nucleus of the vagus nerve is motor and sensory. The dorsal motor nucleus of the vagus lies along the lateral side of the nucleus of the hypoglossal nerve. From the cells of this nucleus arise the different fibers of the vagus and glossopharyngeal nerves which innervate

5. Ranson, S. W.: Anatomy of the Nervous System, Philadelphia, W. B. Saunders Company, 1928.

smooth muscle and glandular tissue of the esophagus, the stomach, the intestine, the bronchial tree, the trachea and the heart.

Through the dorsal sensory nucleus the vagus and glossopharyngeal nerves receive sensory impressions from the soft palate, the posterior third of the tongue, the pharynx, the epiglottis, the larynx, the trachea, the respiratory mucosa, the esophagus, the stomach, etc.

Wallenberg,[6] in 1895, was the first to give a detailed description of the symptoms of this syndrome, and since then numerous cases of this kind have been reported.[7] The syndrome is sometimes known as the Wallenberg syndrome.

I may be pardoned if I digress for a moment to mention some of the other important syndromes related to this area of the medulla oblongata. I am prompted by the presence of interesting laryngologic symptoms associated with these neurologic syndromes, and it is essential that they be properly evaluated.

Tilney and Riley [4] mentioned and interpreted the following syndromes:

A. Syndrome of the nucleus ambiguus and spinothalamic tract, or syndrome of Avellis.

1. Ipsilateral paralysis of the vocal cord and soft palate.
2. Contralateral loss of pain and temperature (somatic) sensibility in the leg, trunk, arm and neck.

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6. Wallenberg, A.: Acute Bulbaraffection, Arch. f. Psychiat. **27**:504, 1895.

7. Anderson, A. C.; Lockhart, R. D., and Souter, W. C.: The Lateral Syndrome of the Medulla, Brain **54**:46, 1931. Beevor, C. E.: The Cerebral Arterial Supply, ibid. **30**:403, 1907. Deggle, H. F., and Stopford, J. S. B.: Posterior Inferior Cerebellar and Vertebral Thrombosis, Lancet **1**:1214, 1935. Foix, C.; Hillemand, P., and Schalit, I.: Sur le syndrome latéral du bulbe et l'irrigation du bulbe supérieur, Rev. neurol. **1**:160, 1925. Gillis, A. C.: Occlusion of the Posterior Inferior Cerebellar Artery, J. A. M. A. **63**:1550 (Oct. 31) 1914. Hall, A. J., and Eaves, E. C.: Posterior Inferior Cerebellar Artery Thrombosis with Unusual Features, Lancet **2**:975, 1934. Hun, H.: Analgesia, Thermic Anaesthesia and Ataxia, Resulting from Foci of Softening in Medulla Oblongata and Cerebellum Due to Occlusion of the Left Posterior Inferior Cerebellar Artery: A Study of the Course of the Sensory and Coordinating Tracts in the Medulla Oblongata, New York M. J. **65**:513, 1897. Magnusson, H. J.: Thrombosis of the Posterior Inferior Cerebellar Artery, Lancet **1**:666, 1938. Mollison, W. M.: Dysphagia Due to Pharyngeal Paralysis, J. Laryng. & Otol. **43**:769, 1928. Sandler, S. A.: The Syndrome of the Superior Cerebellar Artery, J. Nerv. & Ment. Dis. **82**:263, 1935. Shellshear, J. L.: Blood Supply of the Dentate Nucleus of the Cerebellum, Lancet **1**:1046, 1922. Spiller, W. G.: Symptom-Complex of Thrombosis of the Posterior Inferior Cerebellar Artery, J. Nerv. & Ment. Dis. **34**:365, 1908. Stopford, J. S. B.: The Arteries of the Pons and Medulla, J. Anat. **50**:131, 1915; **51**:252, 1915. Wilson, G., and Winkelman, N. W.: Occlusion of the Posterior Inferior Cerebellar Artery, J. Nerv. & Ment. Dis. **65**:125, 1927.

3. Retention of all other types of somatic sensation in the areas showing defects in pain and temperature sensibility.
4. Absence of any other sensory or motor disturbance in the body.
5. Presence of Horner's syndrome if the lesion extends dorsally to involve the pupillary center of the medulla.

B. Syndrome of the nucleus ambiguus and nucleus hypoglossus, or syndrome of Tapia.

1. Ipsilateral paralysis of the vocal cord and soft palate.
2. Ipsilateral paralysis, with atrophy of the tongue.
3. Absence of all other motor and of sensory symptoms.

C. Syndrome of the nucleus ambiguus and nucleus accessorius, or syndrome of Schmidt.

1. Ipsilateral paralysis of the vocal cord and soft palate.
2. Ipsilateral paralysis of the sternocleidomastoid and trapezius muscles, producing inability to turn the head, so that the chin points to the side of the lesion (cephalogyric paralysis).
3. Absence of all other splanchnic motor paralysis.
4. Absence of any somatic motor or sensory disturbances.

D. Syndrome of the vagus-accessory-hypoglossal nerves, or syndrome of Jackson.

1. Ipsilateral paralysis of the soft palate.
2. Ipsilateral paralysis of the larynx.
3. Ipsilateral paralysis of the sternocleidomastoid and trapezius muscles (cephalogyric paralysis).
4. Ipsilateral paralysis of the tongue, accompanied by atrophy.
5. Absence of all other splanchnic motor or of any somatic motor or sensory disturbances.

REPORT OF A CASE

J. L., aged 49, drove from New York city to Miami Beach, Fla., for his vacation about the middle of December 1940, while suffering from a low grade infection of the upper respiratory tract. After a few days, while swimming, he complained of pain in the right shoulder, and a diagnosis of sprained ligament was made. The right arm was strapped, and diathermy treatments were given. The pain, however, persisted and increased in intensity to such a degree that the patient suggested hospitalization. He was admitted to St. Francis Hospital.

A roentgenogram of the shoulder revealed nothing significant. However, three days after admission a swelling appeared in the clavicular region concomitant with intermittent elevation of temperature. This swelling gradually extended to the right side of the neck, with symptoms of pressure on adjacent structures, such

as slight difficulty in swallowing and a dry cough. Consultation with respect to the ears, nose and throat revealed nothing significant. With conservative therapy, the swelling of the neck subsided and the temperature became normal.

However, three weeks from the onset of the illness, and directly after the diathermy treatments, pyemia developed, with embolization of the lungs and bronchopneumonia; later the embolic process spread to involve the spleen, then the liver, producing mild jaundice, and then the kidneys, with appearance of red blood cells in the urine.

Flaccid paralysis of the left arm and leg, paralysis of the left side of the face, of central type, and paralysis of the left vocal cord developed. The patient began to cough, with profuse productivity and choking; food was vomited, and he became incontinent of urine and feces and passed into muttering delirium. He was irrational, chiefly about business affairs. A blood transfusion was given, and the patient rallied a little. However, the condition soon became serious again.

The patient was then removed to the Miami Beach Hospital, and Dr. Henry Barrett was consulted with regard to hemoirradiation. The response to this therapy was so spectacular that the procedure was repeated on two other occasions with such rallying effect as to enable the patient to make the trip back to New York.

On admission to Manhattan Eye, Ear and Throat Hospital, on Jan. 31, 1941, he was thin and emaciated and was suffering from cachexia and malnutrition. He was totally confused and disoriented and talked incessantly and incoherently. He did not know where or who he was.

Examination of the ears, nose and throat revealed that the drum membranes were normal and hearing was intact. The sense of smell was unimpaired; there was low grade chronic ethmoiditis, and the nasopharynx, mouth, larynx and trachea were filled with small milky curds of secretion. The mobility of the tongue was not involved. There was paralysis of the left side of the soft palate. On phonation the right side of the soft palate contracted upward. Examination of the larynx was easily performed, there being apparently complete loss of sensation in the soft palate and pharynx. The left vocal cord was completely paralyzed, which accounted for the hoarseness. The patient experienced great difficulty in swallowing, especially fluids.

The patient past pointed spontaneously to the left and had a feeling of falling to the left. Nystagmus was present from the first day and persisted up to two days before his release from the hospital. It became more pronounced during the course of the illness. This nystagmus was constant and was present in all positions of the eye and was not associated with dizziness. At one time it was rotary and at another horizontal, the direction differing from time to time. Five cubic centimeters of ice cold water was inserted into the left ear, and immediately there was a marked change in the degree and intensity of the nystagmus. Rotary nystagmus to the right was elicited immediately and lasted for at least two minutes. Marked dizziness was associated with this induced nystagmus, which was normal in direction but hyperactive in degree, time and amplitude.

The eyes were normal except for superficial, punctate keratitis, with exfoliation of the corneal epithelium and xerosis and complete anesthesia of the conjunctiva and cornea of the left eye. Horner's syndrome was not present. The right eye showed complete anesthesia but no other involvement. These observations indicate that the cause of the ocular lesions probably was cachexia and malnutrition associated with a lesion of the central nervous system, with the production of

neuropathic keratitis in the left eye. The possibility of an abscess of the fronto-parietal lobe on the right side in an embolic process was considered. This was immediately ruled out because the veins of the fundi, especially the right, would have been engorged if this had been the case.

Dr. Clarence Hare found the following neurologic signs: marked ataxia of the upper and lower extremities on the left side; loss of pain sensation on the right side of the body up to the second cervical dermatome and some degree of lessened sensibility over the left side of the face; very slight diminution in touch and vibratory sense on the right side; normal muscle, joint and tendon senses; suppression of all reflexes (tendon), and absence of a definite Babinski sign. The left arm and leg were weak. The impression was that of thrombosis of the left posterior inferior cerebellar artery, possibly of an infectious nature.

The sedimentation time showed a diagonal curve of forty-five minutes, and the sedimentation index was 22 mm. Lumbar puncture, chemical studies of the blood and urinalysis gave normal results. Culture of the sputum yielded a mixed growth of Leptothrix, nonhemolytic streptococci and Monilia albicans.

The patient was given a high caloric diet and daily intramuscular injections of vitamins A, B and C for relief of the cachexia and malnutrition. Juices of all kinds were forcibly administered. The coagulation time was normal, but the prothrombin time was forty-five seconds. Vitamin K was then given. When the prothrombin time became normal, administration of this vitamin was discontinued.

A slight rise in temperature occurred every third day, but a blood smear was negative for malaria organisms. There was no evidence of aleukemic leukemia, Hodgkin's disease, subacute bacterial endocarditis, typhoid or paratyphoid. The patient was progressing well except for a low grade fever in the evening of 100 F. and a pulse rate of 100 to 140 per minute. The sedimentation time was gradually becoming normal. He was now able to swallow and was able to coordinate the movements of the upper and lower extremities. The paralysis of the soft palate and vocal cord was gone. Tone in the upper and lower extremities was gradually becoming stronger. Sensation in the soft palate and pharynx was returning, for on laryngeal examination he began to gag. There was now a paresthesia over the left side of the face.

On the forty-second day in the hospital the temperature rose to 102 F., and the pulse rate from 100 to 138, with marked pains in the left inguinal region. The sedimentation index and sedimentation time which two weeks ago had been 17 mm. and fifty minutes, respectively, now were 24 mm. and thirty minutes. The pain was not relieved by local application of heat but was helped by sedatives. For three days the patient complained of excruciating pain in the inguinal region, which was aggravated every time he moved his left leg. Hemoirradiation was then given by Dr. Henry Barrett. The pain completely disappeared the next day, and the temperature fell by lysis.

On the forty-seventh day, at his request, the patient was released from the hospital, with a temperature of 101 F. and a pulse rate of 108 per minute. The respiration and blood pressure were normal throughout the course of the illness. The patient made slow progress at home, without remission, and within six months was fully recovered from his illness.

The swelling in the right side of the neck following a low grade infection of the upper respiratory tract can reasonably be explained on the

assumption that a septic mural thrombus developed in the internal jugular vein. The manipulation associated with swimming and the diathermy, I believe, aggravated this protective thrombus, with dissemination of septic emboli to the lungs, spleen, liver, kidneys, frontoparietal area, dorsal lateral aspect of the medulla oblongata and, finally, left inguinal region. Short wave diathermy, as usually applied with large size electrodes or an inductance coil, results in spreading of the lines of force over all adjacent structures and in a good deal of wasted energy. This must be considered in treating heat-sensitive patients and in applying the therapy to such locations as the head or the neck, where unnecessary heating of non-affected parts is to be avoided (Kovacs [8]).

The frontoparietal area of the brain is supplied by the terminal branches of the internal carotid artery. The ascending parietal branch of the middle cerebral artery supplies the precentral convolution, and the frontal branches of the anterior cerebral artery supply the frontal lobe. Occlusion of the main trunk of the anterior and middle cerebral arteries only can explain the pathologic changes involved. If this occlusion had been complete, softening would have occurred within six hours and there would have been permanent damage to this area. The fact that the patient recovered from this frontoparietal syndrome indicates that the occlusion was only partial and that softening did not occur.

The posterior inferior cerebellar artery is the longest branch of the vertebral artery and is rather a tortuous vessel of no great caliber. Its length and tortuosity are the principal reasons that this branch is involved more often than the other branches in this neighborhood.

The ipsilateral hemiasynergia and hemiataxia of the upper and lower extremities are due to the involvement of the ventral and dorsal spinocerebellar tracts. The ipsilateral hemifacial anesthesia, including all types of sensibility, results from a lesion of the descending tract of the trigeminal nerve. The contralateral loss of pain and temperature in the arm, leg and neck is due to the involvement of the lateral spinothalamic tract, which conveys sensory impulses from the opposite side of the body. The paralysis of the left side of the soft palate and the left vocal cord is explained by the lesion of the left nucleus ambiguus. The central nystagmus, which was constant for forty-five days, is accounted for by the irritation of one of the vestibular nuclei, namely, the triangularis. The loss of sensation over the soft palate and the pharynx is explained by the involvement of the sensory fibers of the glossopharyngeal nerve.

The heart has a triple nerve supply: an intrinsic innervation from the ganglions of the heart; sympathetic fibers and fibers from the vagus nerve. The vagus acts as an inhibitor of cardiac action. The center

8. Kovacs, R.: Evaluation of Short Wave Diathermy, Brit. J. Phys. Med. 3:5, 1940.

for the inhibitory control of the cardiac cycle is situated in the dorsal motor nucleus of the vagus nerve. This may explain why the pulse in this case continued to be rapid, in spite of the fact that the paralysis of the soft palate and vocal cord returned to normal. The focus of infection was still present, which explains the low grade fever and the high sedimentation rate, and probably the rapid pulse. The low grade fever throughout the course of the illness is accounted for by the persistence of the thrombus in the internal jugular vein, which after forty-five days of quiescence started on another rampage, with involvement of the left inguinal region.

The occlusion of the posterior inferior cerebellar artery was not complete in this case. Time is an important factor in determination of the degree of occlusion. Ataxia and asynergia are signs of cerebellar involvement. These disappeared soon, for the posterior inferior cerebellar artery is not an end artery to the cerebellum, which is known to be further supplied by the anterior inferior cerebellar and the superior cerebellar branch of the basilar artery. The posterior inferior cerebellar artery is, however, an end artery to the dorsolateral region of the medulla oblongata, which gives rise to this syndrome. The fact that the paralysis of the soft palate and the vocal cord disappeared, that there was no more disturbance in deglutition, that sensation of the soft palate and pharynx returned and that paresthesia of the left side of the face was present and sensation of pain was returning to the right side of the body speaks for partial occlusion in this case.

ULTRAVIOLET IRRADIATION OF THE BLOOD

The method employed in irradiating the blood in this case was developed by Dr. E. K. Knott and first tried on a human subject in 1928. Several thousand persons have received this form of therapy.

The technic in brief is as follows (Barrett[9]): "One and twenty-five hundredths cubic centimeters of blood per pound (0.5 Kg.) of body weight is removed by means of a transfusion needle, mixed with a 2.5 per cent solution of sodium citrate and returned immediately to the patient via the Knott hemoirradiator, which consists of a pump, a mechanism to vary the irradiation time and an irradiation chamber. The source of ultraviolet radiation is a water-cooled type of quartz–mercury vapor lamp. The average dose of radiation given the blood is one second per cubic centimeter. The number of treatments and the interval between treatments depend on the nature and the severity of the disease. This patient received 3 treatments in ten days at Miami Beach Hospital and

9. Barrett, H. A.: Hemoirradiation by Means of Ultraviolet Spectral Energy, personal communication to the author.

was then brought to the Manhattan Eye, Ear and Throat Hospital, where he received 2 hemoirradiations during his stay of seven weeks. The number of treatments given 250 patients averaged a little over 2, the greatest number given any one patient being 8.

Except for an occasional reaction to the citrate solution, I have noticed no untoward effects. The incidence of these reactions is 1 in 50 irradiations. Deleterious effects on the blood elements have not been seen in any of the patients I have treated. As a rule there is early improvement in the blood picture. Disappearance of toxic manifestations associated with toxemia is generally prompt, definite improvement frequently being seen in a few hours. Return of appetite is commonly observed even as early as twelve to twenty-four hours. Attention is called to the prompt relief of pain in the left inguinal region in this case when other measures of relief had failed. Such an effect is often seen.

The effects of hemoirradiation by the Knott method may be summarized as follows: (1) early diminution of toxemia; (2) improvement in the blood picture; (3) fall in temperature by lysis; (4) relief of pain; (5) improvement in the circulation, and (6) early return of appetite. The diminished toxemia will of course account for the improvement in the blood picture, as well as the fall in temperature and the general improvement in the patient's condition.

Elsewhere I have discussed the possible modus operandi of hemoirradiation, but I am frank to admit that at present one possesses little knowledge of the precise manner in which ultraviolet energy exerts its beneficial action, not only as it is used in the Knott method but as it is employed conventionally. Its use, like that of all medical agents and most surgical procedures, is empiric. The technic employed in this new method of administering ultraviolet energy has been developed by adherence to the time-honored principle of "trial and error."

CONCLUSION

The syndrome of the posterior inferior cerebellar artery is a recognized neurologic entity. The presentation of this syndrome to the otolaryngologist is permissible, as a careful search in the otolaryngologic literature reveals nothing on this subject. The diagnosis in cases of this condition calls for comprehensive knowledge of the anatomy of these parts and evaluation of the innervation of the cranial nerves and tracts involved. While the otolaryngologist is not directly responsible for the diagnosis of this condition, he is, on the other hand, fully responsible for the proper interpretation and evaluation of those otolaryngologic symptoms which fit into the pattern of this neurologic syndrome.

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CONGENITAL PREAURICULAR CYSTS AND FISTULAS

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A congenital pit or sinus involving the ascending limb of the helix of the ear (fig. 1 *a*) or the preauricular region is of little importance unless it gives rise to an offensive discharge or forms a retention cyst (fig. 1 *b*). When infected, such a lesion is of considerable clinical significance, since drainage, extension, scarring and disfigurement are the common sequelae. Infection also may result in a secondary preauricular lesion, which so dominates the clinical picture that the underlying fistula itself may be overlooked entirely.

Differences in nomenclature and in methods of treatment and contradictory theories advanced to explain the origin of such pits and sinuses lend confusion to a study of the literature on this subject. These embryonic structures are known by a variety of names, such as "sinus preauricularis congenita," "branchial fistula in the external ear," "fistula auris congenita," "helical fistula" and "preauricular fistula."

Although these sinuses, or fistulas, are unquestionably of congenital origin, there is no certainty as to their mode of development. Many observers have assumed that they arise from the first branchial cleft; however, Ballantyne [1] came to the conclusion that there are at least two difficulties which prevent the acceptance of this view: first, the freedom of the middle ear from participation in the deformity and, second, the position of the fistulas. Stammers,[2] in his report of 2 cases with a review of the literature, concluded that these fistulas are not remnants of the first branchial cleft but result from an aberrant coalescence of the six tubercles which are destined to form the pinna. Ladd and Gross [3] expressed the opinion that the ear is formed from six tubercles which

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1. Ballantyne, J. W.: Manual of Antenatal Pathology and Hygiene: The Embryo, Edinburgh, W. Green & Son, Ltd., 1940, vol. 2.

2. Stammers, F. A. R.: Pre-Auricular Fistulae, Brit. J. Surg. **14**:359-363 (Oct.) 1926.

3. Ladd, W. E., and Gross, R. E.: Congenital Branchiogenic Anomalies: Report of Eighty-Two Cases, Am. J. Surg. **39**:234-248 (Feb.) 1938.

are derived from the first and second branchial arches. They said that preauricular fistulas result from improper fusion of the three nubbins of the first arch, which form the helix and tragus of the ear. As a result of a study of the development, the anomalies and the nerve supply of the external ear, Jones and Wen [4] maintained that the pinna is essentially a hyoid derivative and that the mandibular contribution is represented only by the tragus and the anterior part of the external auditory meatus. Consequently, they concluded that preauricular fistulas lie along the line of the first pharyngeal depression.

It is apparent from this brief summary that there is no certainty as to the exact origin of these fistulas. Although the theories mentioned are of interest from an embryologic standpoint, they are of little practical

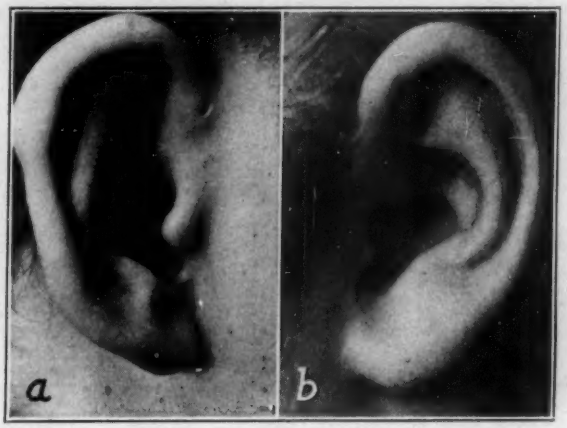

Fig. 1.—(*a*) Common site and appearance of an uninfected preauricular fistula. (*b*) Infected retention cyst anterior to the opening of a preauricular fistula.

significance clinically. Of more importance is the fact that the hereditary nature and transmission of this condition have been observed by many writers, and considerable evidence has been accumulated to put this fact on firm ground (fig. 2). For example, Schüller,[5] who collected records of 70 persons from four generations of a direct line of descent, found that 26 of the 70 had auricular fistula.

Although many asymptomatic preauricular pits and sinuses are overlooked, there is no doubt that this condition is comparatively uncommon.

4. Jones, F., and Wen, I. C.: The Development of the External Ear, J. Anat. **68**:525-533 (July) 1934.

5. Schüller, J.: Zur Vererbung der Fistula auric. bzw. auriculae congenita; zugleich ein klinischer Beitrag, München. med. Wchnschr. **76**:160-162 (Jan. 25) 1929.

As evidence of its rather rare incidence, Urbantschitsch [6] found 12 instances of fistula among 2,000 persons, and Onidi,[7] 48 instances among 3,200 persons. Congdon, Rowhanavongse and Varamisara,[8] who made a study of Oriental races, reported an incidence of 4 per cent in some racial stocks and 6 per cent in others. Selkirk,[9] in the routine physical examination of 3,600 white school children, found 34 instances of this condition.

In a period of twenty years, only 16 patients came to the Mayo Clinic for treatment of preauricular pits or sinuses, and only 13 of these received surgical therapy. Although many other patients with asymptomatic preauricular fistulas were seen at the clinic in this time, this study has been confined to these 16. When the histories were reviewed, it was

Fig. 2.—Infected, discharging preauricular fistulas in father (*a*) and son (*b*). Scarring of the face surrounding the opening is the result of infection and previous treatment.

discovered that all but one of the fistulous tracts were infected at the time of examination. The majority of the patients had received previous treatment. In more than half of the cases (fig. 2) the use of phenol or other cauterizing agents, incision for drainage, the application of salves,

6. Urbantschitsch, V.: Concerning the So-Called Fistula Auris Congenita: Anomaly of Formation, Edinburgh M. J. **23**:690-695 (Feb.) 1878.

7. Onidi, L.: Ueber kongenitale Ohrfisteln, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **102**:128-136, 1918.

8. Congdon, E. D.; Rowhanavongse, S., and Varamisara, P.: Human Congenital Auricular and Juxta-Auricular Fossae, Sinuses and Scars Including the So-Called Aural and Auricular Fistulae and the Bearing of Their Anatomy upon the Theories of Their Genesis, Am. J. Anat. **51**:439-464 (Nov.) 1932.

9. Selkirk, T. K.: Fistula Auris Congenita, Am. J. Dis. Child. **49**:431-447 (Feb.) 1935.

irradiation or neglect had resulted in much scarring about the tragus or in the preauricular region. The incidence according to sex was equally divided, and the fistulas or associated cysts were noted at all ages from birth to 38 years. In half of the cases the condition was bilateral; in the other half it was unilateral. It was interesting to find that the onset of infection in the fistulas had occurred most frequently in early childhood and that in 4, or 25 per cent, of the 16 cases 2 or more members of the family had had auricular fistulas.

Preauricular fistulas vary greatly in their gross anatomic structure. At one extreme is the simple tubular sinus which is only a few millimeters in length and lined with squamous epithelium. At the other extreme is the complex sinus, possessing one or more offshoots that diverge in any direction.

It is not uncommon to encounter complex sinuses which exhibit nothing more than a pit externally but which infiltrate deeply into the preauricular region and extend into the tissues of the tragus and helix or even to the pharyngeal wall. Many physicians apparently are not aware that the structure of many preauricular fistulas is most intricate, which makes surgical removal a difficult task. The external appearance of an asymptomatic preauricular sinus or fistula amounts to nothing more than a simple pit. However, with obstruction of this orifice the tract may develop into a typical cystic tumor (fig. 1 *b*). Infection is the complication which produces the greatest changes in the outward appearance of these fistulas. Chronic infection causes a more or less continuous formation of purulent material, which is discharged externally. Under such circumstances, the skin overlying the fistula undergoes much excoriation, eventual ulceration and ultimate scarring.

Histologic studies of the tissue removed at the time of operation in our series of cases revealed tracts or cystic cavities lined with squamous epithelium associated with subepithelial fibrosis and inflammation (fig. 3). In several instances, owing to infection or to mechanical distention of the cavity or tract, the epithelial lining was found to be entirely destroyed. The degree of fibrosis and loss of epithelial structure in these fistulas was found to be proportional to the extent and duration of the infection and to the amount of previous treatment. In an instance in which a solution of phenol had been used several times, no epithelium-lined tract could be found, although many hair follicles were present in a mass of inflammatory and scar tissue. No suggestion of a malignant change was noted in any of the specimens which we examined; however, it is possible that a malignant tumor could develop in a preauricular fistula, and Bell [10] has stated that carcinoma occasionally arises in a branchial cyst or fistula.

10. Bell, E. T.: Text-Book of Pathology, ed. 3, Philadelphia, Lea & Febiger, 1938, p. 634.

The successful treatment of preauricular fistula, or cyst, is based on two methods: (1) the complete removal of the cystic sac or fistulous tract and (2) the destruction of the epithelial or fibrous lining. The first method refers to surgical extirpation, in which the entire sac or fistulous tract is dissected free from the surrounding tissues. It is advantageous to remove an elliptic piece of skin surrounding the orifice of the sinus before attempting excision of the tract itself. Our studies have shown that the removal of simple preauricular fistulas by sharp dissection is the ideal method of therapy and insures a minimal amount of postoperative scarring. However, the surgical removal of the sinus is sometimes difficult because of poor visualization of the tract.

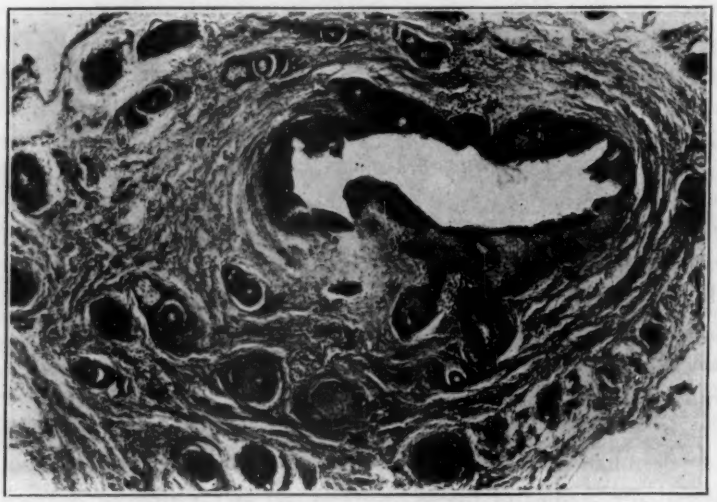

Fig. 3.—Cross section of preauricular fistula, showing squamous epithelial lining surrounded by multiple hair follicles and associated with subepithelial inflammation (× 50).

The use of a fine purse string suture encircling the orifice of the fistula, as recommended by Love[11] for thyroglossal cysts, is not applicable to fistulas anterior to the tragus. The extent of the fistulous tract may be determined easily by the injection of methylthionine chloride (methylene blue) or other coloring substances in the manner outlined by Baumgartner.[12] The use of a probe inserted in the fistulous tract is also very helpful. The making of roentgenographic studies after

11. Love, R. J. M.: A Simple Method of Dealing with Congenital Fistulae, Lancet **2**:122 (July 20) 1929.

12. Baumgartner, C. J.: Branchial and Thyroglossal Duct Cysts and Fistulas in Children, Surg., Gynec. & Obst. **56**:948-955 (May) 1933.

the injection of an opaque medium into the sinus has been recommended by Wangensteen.[13]

Owing to the branching of some of these fistulous tracts, their surgical removal by sharp dissection is often extremely difficult and sometimes impossible. It is this fact which has led to the use of the second form of treatment, that is, destruction of the epithelial or fibrous lining, either by the use of sclerosing solutions or by surgical diathermy. The injection of sclerosing agents is rarely effective, because the solution does not reach the necessary depths, nor does it penetrate the wall sufficiently to obliterate the fistula completely. In our group of cases the highest incidence of recurrence was observed in those cases in which sclerosing solutions were used, either at the clinic or before the patients came to the clinic.

Surgical diathermy is probably the most effective method of treating complex preauricular fistulas. Moreover, electrocoagulation is particularly useful in cases in which patients have received much previous treatment. In these cases, the residual scar tissue can be excised and the underlying tissues thoroughly destroyed by diathermy. It has become apparent to us that a method recently described by Havens[14] no doubt offers the best assurance of a permanent cure, particularly in cases in which there are multiple tracts.

Havens said:

> The sinus is first filled with methylthionine chloride (methylene blue) in order to stain its lining. For this I use an ordinary 2 cc. syringe, breaking off the needle where it emerges from the conical portion of the butt. The cone-shaped tip thus provided is fitted snugly into the opening of the sinus, and the injection is made to distend the entire tract. Following this, the electric scalpel (so-called radio knife) is used to lay open the sinus and all its ramifications. Because the electric scalpel eliminates bleeding the stained sinal tracts can be seen and accurately followed. After all have been laid open, the entire region is electrocoagulated sufficiently to destroy the epithelial lining. The resulting wound heals by granulation in four to six weeks, and unless the region involved has been unusually large the resulting scar is not conspicuous.

102 Second Avenue South West.

13. Wangensteen, O. H.: Differentiation of Branchial from Other Cervical Cysts by X-Ray Examination, Ann. Surg. **93**:790-792, 1931.

14. Havens, F. Z.: Congenital Branchiogenic Preauricular Sinus: A Note Regarding Its Treatment, Arch. Otolaryng. **29**:985-986 (June) 1939.

Case Reports

TRANSSEPTAL REMOVAL OF CHONDROMA REPLACING SPHENOID SINUS AND SELLA

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The comparative rarity of enchondromas of the sphenoid sinus is the primary reason for this report. It may be also of interest that such extensive involvement as occurred in this case was relieved by a relatively simple operation. The ease of the procedure suggests that perhaps more tumors of the region of the sella might be treated by a rhinologic approach.

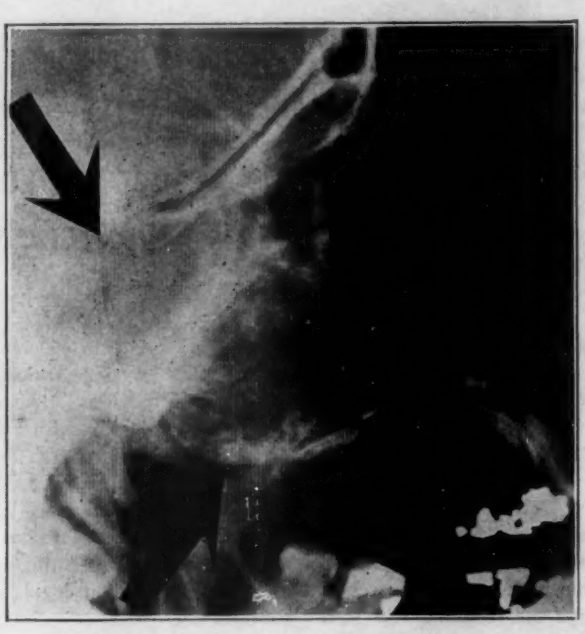

Fig. 1.—Roentgenogram, showing a tumor mass involving the sella, the sphenoid sinus and the septum.

REPORT OF A CASE

History.—Miss M. F., a retired school teacher aged 76, presented herself on June 19, 1939 with the following complaints: headaches, becoming progressively worse for two years and almost daily now; nasal blocking for seven years but becoming progressively worse for the last two years; progressive loss of vision in the left eye for two years, so that this eye now had only light perception, and several attacks of nasal hemorrhage in the past four years. The headache became worse at night or in the early morning or on lying down but was noticeably improved by sitting up. There had been some tinnitus in the left ear for many years. Her weight had decreased 35 pounds (15.9 Kg.) in seven years, and she had been told that at times her blood pressure was too high or too low.

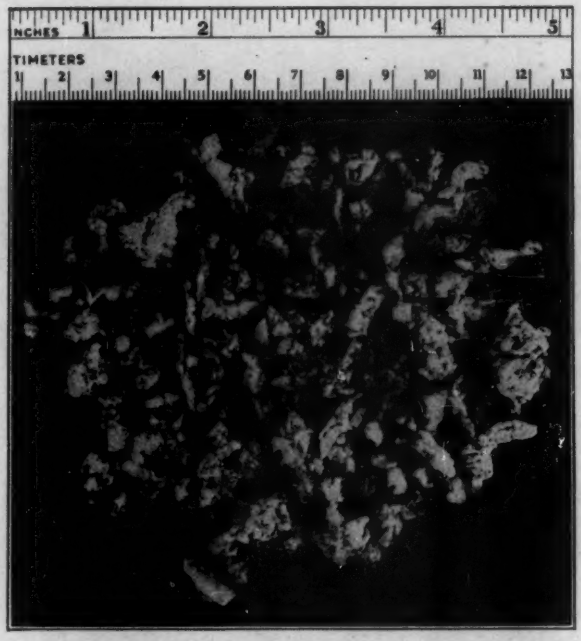

Fig. 2.—Shavings of the chondroma removed.

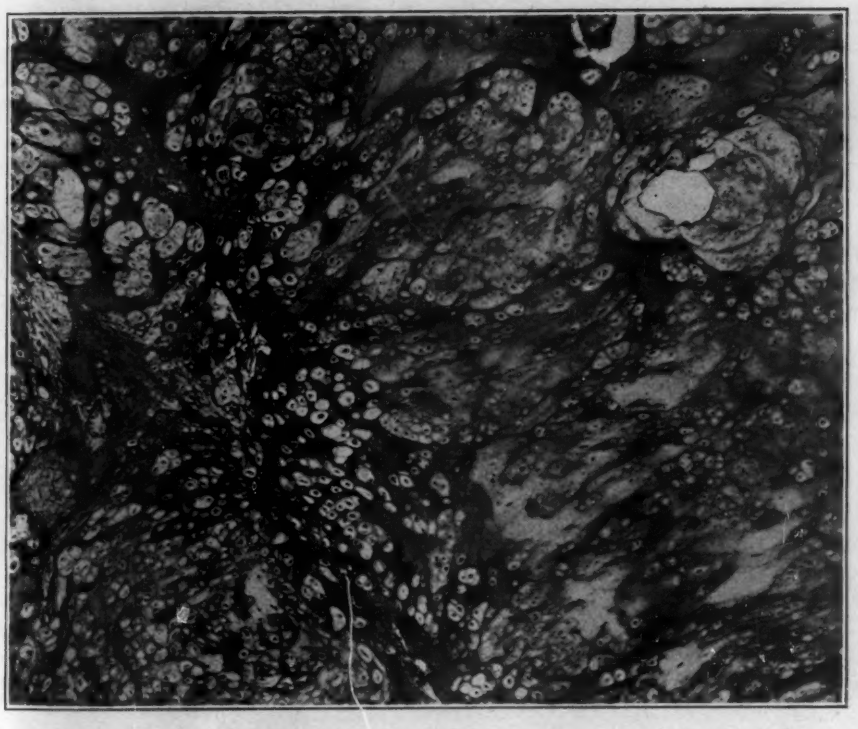

Fig. 3.—Section of an atypical bizarre benign chondroma, consisting of chondroblasts and chondrocytes in a hyaline matrix.

Examination.—The mucous membrane was very pale and boggy, with considerable deviation of the septum. There was a firm round hard symmetric mass at the posterior end of the septum, with almost complete blocking. Vision in the right eye was 20/50, but improved to almost 20/20 with correction; the eyeground was normal and the temporal field slightly contracted. The left eye had only light perception, with the temporal half of the field blind. The left optic disk was pale (optic atrophy). The blood pressure was 150 systolic and 78 diastolic; the Kahn reaction of the blood was negative, and the white cell count was 12,500.

The report of the roentgenographic examination (Dr. T. C. Childs) follows: "The sella turcica, particularly the posterior clinoid process, was eroded, with complete destruction of the roof of the sphenoid sinus. The tumor most likely to produce such changes is a chromophobe adenoma."

Biopsy (Dr. E. L. Benjamin, pathologist to the Evanston Hospital).—"The neoplasm was formed by a pale homogeneous matrix in which were dispensed slightly irregular formations of chondroblasts and chondrocytes. Small areas of ossification were present, associated with rather pronounced cellular activity. The diagnosis was ossifying chondroma."

Operation (T. C. G.).—The tumor was removed at the Evanston Hospital on July 18, 1939, with the use of local anesthesia. The mass replaced the posterior half of the septum, obliterating the sphenoid sinus and invading the body of the sphenoid bone, pressing shallow bone down into the nasopharynx high up on the left side and merging somewhat gradually into the septum below. The tumor had a hard cortex, with some areas which were rather soft and cartilaginous. Through a left septal incision the midportion of the nasal septum was resected. After primary opening with biting forceps, the center of the tumor was curetted out. Then, by cutting forward, chiefly with a Coakley curet, the mass was rather easily enucleated in thin slices. Gray glistening dura, pulsating and intact, was exposed over the whole central area of the sphenoid. An iodoform gauze basket pack was left in the operative wound.

Postoperative Course.—The patient was given sulfanilamide for the first five days after operation. Slight mental confusion while in the hospital was attributed by an internist to her age and a hypotensive phase of hypertension. She left the hospital ten days after operation. Two months later there was some crusting of the nostrils, but a year later the nasal wound was smooth and clean. The right eye still had 20/25 vision and a good field, and the left eye still had light perception in the nasal half. The patient is still in good health, twenty-seven months after operation.

COMMENT

Ringertz,[1] in his study of 391 cases of tumor of the nasal and paranasal cavities, found only 2 instances of enchondroma. In 1 of the cases there had been no recurrence four years after one operation and in the other none four years after the third operation. He expressed the belief that this tumor arises from remnants of the embryonal cartilaginous skeleton which escape resorption during enchondral ossification. The tumor consists of relatively mature cartilage and myxomatous tissue, and the cartilage is often very cellular. Ringertz found in the literature reports

1. Ringertz, N.: Pathology of Malignant Tumors Arising in Nasal and Paranasal Cavities and Maxilla, Acta oto-laryng., 1938, supp. 27, pp. 1-405.

of about 70 tumors containing chiefly cartilage in the nose and sinuses, and of these about 50 appeared to be chondromas. Most of these arose from the ethmoid or the sphenoid bone, both of which are preformed in cartilage. The tumor may vary from the size of a walnut to that of a hen's egg, or even more. It is firm, smooth, elastic and irregularly lobulated. The cut surface is gray-white to red, is shiny and often shows deposits of calcium. It grows expansively, erodes by pressure and destroys bone and may thus invade the lamina cribrosa and the cranium. Most of the tumors were discovered fairly early in life, and in no case was the patient as old as ours. Hickey [2] reported a case of chondroma of the ethmoid, with a review of the literature.

Ewing [3] stated that the structure of the chondroma reproduces that of the various normal types of cartilage, chiefly the hyaline variety. The matrix of the chondroma is usually hyaline, but may be fibrillated or contain elastic fibrils.

Rhinologists have played an important part in the pioneer development of pituitary surgery and have reported some of the best results. Hirsch [4] proposed the transphenoid operation on the pituitary gland in 1910, and similar surgical procedures were described in articles by Schloffer [5] (1906), Kanavel [6] (1909), Halstead [7] (1910) and Cushing [8] (1912). Cushing was enthusiastic about the method in the early years but later regarded the transfrontal approach as preferable. Frazier [9] (1924) reported 18 consecutive cases of primary intrasellar lesions removed by transphenoid hypophysectomy, with satisfactory results and no operative deaths. Henderson [10] (1939) reviewed the results in 338 cases of pituitary adenoma operated on by Dr. Harvey Cushing over the previous twenty years. Nager [11] (1940) reported on 47 transphenoid operations on 39 patients; 43 of these were done by an external ethmosphenoid approach (as described by Chiari, in 1911) and 4 through a palatine incision. He recommended the operation as safe for tumors of

2. Hickey, H. L.: Chondroma of Ethmoid, Arch. Otolaryng. **31**:645 (April) 1940.

3. Ewing, J.: Neoplastic Diseases, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

4. Hirsch, O.: Ueber Methoden der operativen Behandlung von Hypophysistumoren auf endonasalem Wege, Arch. f. Laryng. u. Rhin. **24**:129, 1910.

5. Schloffer, H.: Zur Frage der Operationen an der Hypophysis, Beitr. z. klin. Chir. **50**:767, 1906.

6. Kanavel, A. B.: The Removal of Tumors of the Pituitary by an Infranasal Route, J. A. M. A. **53**:1704 (Nov. 20) 1909.

7. Halstead, A. E.: The Operative Treatment of Tumors of the Hypophysis, Surg., Gynec. & Obst. **10**:494, 1910.

8. Cushing, H.: The Pituitary Body and Its Disorders, Philadelphia, J. B. Lippincott Company, 1912.

9. Frazier, C. H.: The Surgery of the Pituitary Body, Arch. Surg. **8**:39 (Jan.) 1924.

10. Henderson, W. R.: The Pituitary Adenomata, Brit. J. Surg. **26**:811-921 (April) 1939.

11. Nager, F. R.: The Paranasal Approach to Intrasellar Tumors, J. Laryng. & Otol. **55**:361 (Aug.) 1940.

this region but warned of the danger of meningitis if hydrocephalus is present. He expressed the opinion that the procedure is indicated chiefly for intrasellar tumors and agreed that the transfrontal route is preferable for suprasellar masses. The presence of central scotoma may point to damage of fibers from the macula in the posterior part of the chiasm, with poor accessibility from above, and he preferred the transphenoid route in cases of this type.

Grant,[12] in a summary of cases of pituitary adenoma, reported on 44 transphenoid operations, with 5 deaths, and 126 transfrontal operations, with 12 deaths. He stated that surgical removal of more than 80 per cent of an adenoma of the hypophysis is generally impossible.

It seems, therefore, that the possibility of rhinologic approach to growths in or around the sella should still have some consideration.

636 Church Street.

12. Grant, F. C.: The Surgical Treatment of Pituitary Adenomas, J. A. M. A. **113**:1279 (Sept. 30) 1939.

REMOVAL OF IMPACTED FOREIGN BODIES FROM THE EXTERNAL AUDITORY CANAL BY THE ENDAURAL APPROACH

BRUCE PROCTOR, M.D., DETROIT

The endaural antauricular approach to the temporal bone offers certain advantages over the classic postauricular approach for the removal of foreign bodies from the external auditory canal and adjacent structures. A more direct exposure in the direction of the axis of the external canal is procured by the endaural approach. Removal of the posterior bony canal wall may be obviated by removing the thinner anterior bony wall in order to free a foreign body which is firmly lodged in the bony external canal. A large portion of the anterior bony wall can be removed without noticeably interfering with the function of the temporomandibular joint. Foreign bodies, such as bullets, embedded in the bony floor of the external canal may be more easily and safely removed by this method. The endaural approach may be a useful adjunct to the armamentarium of the military aural surgeon for the removal of foreign bodies from this region.

In the following case the removal, by the endaural approach, of a bullet embedded in the floor of the bony external canal is recorded.

REPORT OF A CASE

R. A., a woman aged 45, came to the outpatient department of St. Mary's Hospital on Sept. 7, 1941, complaining of headache, pain in the right ear and a profuse foul aural discharge. She had never had any disturbance of the ears until ten years previously, when her husband became intoxicated and discharged a .38 caliber revolver at her. The bullet entered anterior to the tragus and lodged in the firm os tympanicum. An otologist who was consulted at the time advised nonintervention. The ear began to discharge shortly after the accident and continued to do so until the bullet was removed surgically.

Hearing had been greatly impaired in the right ear since the time of the injury. In recent months the patient was disturbed by frequent right-sided headaches and by pain in the right side of the neck. There had been no weakness or paralysis of the facial muscles at the time of the injury.

Examination of the right ear revealed a profuse, fetid otorrhea. A large, rather firm, polypoid mass occluded the external canal at its orifice. Audiometric examination and tuning fork tests revealed complete loss of hearing by air conduction. Roentgenograms of the temporal bone revealed the bullet lying below the floor of the bony external canal and anterior to the mastoid process. The mastoid air cells were well developed, suggesting that there had been no otitis media during early childhood, when pneumatization occurs. The mastoid air cells were clouded, and the bony trabeculae appeared indistinct.

From the Department of Otolaryngology, Wayne University.

The endaural approach was selected to explore the floor of the canal, remove the bullet, inspect the tympanic membrane and cavity and, if necessary, perform a mastoidectomy, an atticomastoidectomy or a tympanomastoidectomy, depending on the condition observed at operation.

With the patient under ether anesthesia, the polyp from the external canal was removed, exposing the bullet embedded in the floor of the bony canal and in the anterior wall of the mastoid process. The overlying portion of the anterior wall of the mastoid was removed to afford a better exposure. The bullet was firmly embedded and could not be elevated from its bed. Since the facial nerve was only 1 or 2 mm. posterior and medial to the bullet, it was decided to curet the bullet away piecemeal rather than remove it in one piece.

1, Stenver's view, showing the bullet lying lateral to the styloid process and below the floor of the external canal.

2, roentgenogram taken with the occiput down, revealing the length and position of the bullet (*B*). Note the pathologic changes in the air cell system of the middle ear, particularly in the infralabyrinthine area (*I*).

On completion of this tedious maneuver, the tympanic cavity was inspected. The external canal medial to the bullet was filled with a thick mass of caseous debris, which had collected behind the aural polyp arising from the floor of the bony external canal. The tympanic cavity was filled with granulation tissue.

Accordingly, a tympanomastoidectomy was performed. On removing the posterior canal wall a fistula was observed between the mastoid tip and the cavity which had been occupied by the bullet. This was probably the portal by which the air cell system of the middle ear had become chronically infected. Had the bullet been removed at the time of the original injury a tympanomastoidectomy might have been avoided.

SUMMARY AND CONCLUSIONS

1. The endaural approach is recommended for the surgical removal of impacted foreign bodies in and about the bony external canal.

2. In cases in which the mastoid air cells are normal this route may make possible the removal of foreign bodies without opening the cells.

3. The endaural approach offers a more direct and better exposure of the contents of the external auditory canal.

4. Soft metallic foreign bodies, such as a bullet, which are firmly embedded in bone may be removed piecemeal by curettage, with less likelihood of injury to adjacent structures.

5. The early removal of foreign bodies from the temporal bone is strongly advocated to avoid permanent damage to the conducting mechanism.

1706 Eaton Tower.

Clinical Notes; New Instruments and Technics

USE OF SULFANILAMIDE AND GAUZE PACKING FOLLOWING INTRANASAL OPERATION

E. Clarence Kern, M.D., Montclair, N. J.

Sulfanilamide and gauze packing has been used with satisfactory results after submucous resection, ethmoidectomy, intranasal antrotomy, etc. When the packing is removed in twenty-four hours, there seems to be less swelling of the mucosa and more rapid healing. The packing does not adhere to the mucosa as does plain gauze, but comes out almost as readily as petrolatum gauze. Furthermore, if it is felt desirable to leave the packing in place a second twenty-four hours, the usual odor resulting from this will be absent and there will be less accumulation of pus in the nose. Although I have never happened to have an intracranial complication following a surgical procedure on the nose, still I feel, rightly or not, a little more secure with this packing when operating in case of a subacute condition, when, occasionally, this is necessary. The drug used is either sulfanilamide or sodium sulfathiazole (the sodium salt of 2-[paraaminobenzenesulfonamido]-thiazole), and the packing is prepared as follows:

A 2 or 3 inch (5 or 7.5 cm.) bandage is used, the width depending on the size of the nasal cavity to be packed. About 2 feet (60 cm.) is opened up and then folded along the midline, forming a V-shaped trough. Gauze is used for this purpose because of the finer mesh. The mesh of the usual hospital gauze is so coarse that the granules of the powder will escape unless it is folded double. The powder, about 4 Gm., is distributed along the fold of the bandage, which is then folded again, making a strip approximately ½ inch (1.27 cm.) wide. The assistant holds the free end so the packing is kept horizontal as it is inserted. In the case of antrotomy, the antrum is loosely filled with the gauze. No reaction or unfavorable result has been observed.

45 Park Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

PLASTIC SURGERY

ROBERT H. IVY, M.D., D.D.S.
AND
HENRY A. MILLER, M.D., D.D.S.
PHILADELPHIA

Surgical problems of the war have emphasized the role of plastic surgery, accelerated the application of new discoveries to this field and stimulated the organized training of surgeons with the armed forces to handle maxillofacial injuries. The large scale incidence of certain types of injuries has resulted in notable advances in management.

ADVANCES IN TREATMENT APPLICABLE IN THE FIELD OF PLASTIC SURGERY

It is generally recognized that infections occur in approximately 3 to 5 per cent of clean surgical wounds. Investigations by Hirshfeld [1] indicate that the sources of contamination may be (1) the air, (2) the hands of the surgeon and assistants or (3) the patient's own skin. Two sources contribute to the bacteria of the air; (*a*) the respiratory tract of members of the operating team and (*b*) the skin, hair and clothing of the operating team and the patient. These can be controlled to a large extent at the source by irradiation of the air and spraying or replacement methods. The part played by the patient's own skin is uncertain. The fact that there is little difference in the reported incidence of wound infection from clinics in which treatment of the skin differs suggests that it is not important.

In treatment of the local wound, Key and Frankel [2] advocate local implantation of sulfanilamide powder not only in contaminated wounds but in clinically clean operative wounds in which infection is particularly to be feared or would be especially undesirable. Commenting on such

1. Hirshfeld, J. W.: Bacterial Contamination of Wounds, from the Air, from the Skin of the Operator, and from the Skin of the Patient, Surg., Gynec. & Obst. **73**:72-79 (July) 1941.

2. Key, J. A., and Frankel, C. J.: The Local Use of Sulfanilamide, Sulfapyridine and Sulfamethylthiazole, Ann. Surg. **113**:284-297 (Feb.) 1941.

a procedure, Key and Burford [3] state that there is no interference with healing and no danger of toxic symptoms and that it is of value in reducing the incidence of wound infections.

For the treatment of infected open wounds, Tompkins, Crook, Haynes and Winters [4] report using pectin as a dressing, with prompt response in clearing up the infection and rapid growth of highly vascular granulation tissue.

The problem of an ideal buried suture material near solution. Aries [5] reports that nylon is well tolerated, has all the good qualities of silk, is stronger and less irritating and does not allow as marked an invasion of its interstices as silk. Localio and Hinton [6] state that cotton is well tolerated also. For best handling it should be boiled ten to twenty minutes and used while wet.

The treatment of burns has been a major problem in the present war. Treatment of the shock is of primary importance, and, according to Tenery and Tenery,[7] far more consideration should be placed on what shall be placed *in* the patient than to what shall be placed *on* him. The intravenous use of plasma in physiologic solution of sodium chloride is very important.

In regard to the external agent used, Wakely [8] finds the application of tannic acid of great value on the trunk, but its use on the face, and particularly on the hands and feet, is strongly contraindicated. Burns of the face are treated with dressings saturated in saline solution, while those of the hand are treated with a saline bath and the use of an aqueous solution of triple dye (2 per cent gentian violet, 1 per cent brilliant green and 0.1 per cent neutral acriflavine). Early skin grafting is undertaken.

Improvement in the coagulating type of treatment of burns is announced by Pickrell,[9] who produces a pliable, translucent, antibacterial eschar by spraying the burn with 3 per cent sulfadiazine (2-[paraamino-benzenesulfonamido]-pyrimidine) in 8 per cent triethanolamine.

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3. Key, J. A., and Burford, T. H.: The Prophylactic Implantation of Sulfanilamide in Clean Operative Wounds for the Reduction of Post-Operative Infections, Surg., Gynec. & Obst. **73**:324-333 (Sept.) 1941.

4. Tompkins, C. A.; Crook, G. W.; Haynes, E., and Winters, M.: Pectin in the Treatment of Various Types of Wounds, Surg., Gynec. & Obst. **72**:222-226 (Feb.) 1941.

5. Aries, L. J.: Experimental Studies with Synthetic Fiber (Nylon) as a Buried Suture, Surgery **9**:51-61 (Jan.) 1941.

6. Localio, S. A., and Hinton, T. W.: The Choice and Use of Cotton for Suture Material, Surg., Gynec. & Obst. **72**:615-618 (March) 1941.

7. Tenery, W. C., and Tenery, J. H.: The Emergency Treatment of Burns, South. Surgeon **10**:759-765 (Oct.) 1941.

8. Wakely, C. P.: The Treatment of War Burns, Surgery **10**:207-233 (Aug.) 1941.

9. Pickrell, K. L.: A New Treatment for Burns, Bull. Johns Hopkins Hosp. **69**:217-221 (Aug.) 1941.

An improved noncoagulating moist method is presented by Bunyan,[10] which consists of employment of a rubber envelop through which passes an irrigating solution of 5 per cent sodium hypochlorite (electrolytically produced).

McIndoe,[11] in his treatment of war burns, finds that sulfanilamide powder sprinkled liberally over the burn as a first aid measure aids in reducing the incidence of subsequent sepsis, and therefore hastens epithelization.

The development in England of centers for management of regional maxillofacial injuries manned by specialist teams has resulted in the undertaking of early skin grafting in cases of burn. There have resulted less pain and care to the patient, a shorter period of hospitalization and far less deformity. Trusler [12] discusses the need of skin grafting procedures as soon as possible and illustrates his methods of grafting. Brown [13] states that the local care of the open wound has for its object the cleaning up of the area as quickly as possible, so that the lost surface may be restored before contractures have occurred and before debilitation and pain have developed beyond control. A graft from one-half to three-quarters the thickness of the whole skin is the most useful in making repair of a raw surface. The deep glands left behind in the derma of the donor site "dedifferentiate" into squamous epithelium and cover the surface in six to eight days. Successive "crops" of this skin may be taken.

DEVELOPMENTS WITHIN THE PROFESSION

Davis,[14] in his presidential address before the Southern Surgical Association, traced the origins and the development of plastic surgery. Koch,[15] in remarking that the surgeon who transplants skin and subcutaneous tissue is indebted to many men whose experience and discoveries have made possible the results which he accepts today as a matter of course, pays tribute in an interesting and entertaining article to these men. Seltzer [16] gives a brief historical review of the attempts

10. Bunyan, J.: The Treatment of Burns and Wounds by the Envelope Method, Brit. M. J. **2**:1-7 (July 5) 1941.

11. McIndoe, A. H.: First Aid Treatment of Burns, Lancet **2**:377-379 (Sept. 27) 1941.

12. Trusler, H. M.: Deformities Due to Burns, Ann. Surg. **113**:1092-1093 (June) 1941.

13. Brown, J. B.: Thick Split Skin Grafts in the Repair of Burns, Surg., Gynec. & Obst. **73**:265-267 (Aug.) 1941.

14. Davis, J. S.: The Story of Plastic Surgery, Ann. Surg. **113**:641-655 (May) 1941.

15. Koch, S. L.: The Transplantation of Skin and Subcutaneous Tissue to the Hand, Surg., Gynec. & Obst. **72**:1-14 (Jan.) 1941.

16. Seltzer, A. P.: The History of Plastic Surgery, Laryngoscope **51**:256-262 (March) 1941.

at fulfilment of William Mayo's observation that "every human being has the divine right to look human."

In discussing the educational needs in the development of plastic surgery, Palmer [17] emphasizes that the greatest stress should be placed on training. This can be accomplished by the establishment of residencies under competent instruction and supervision. Other methods are the providing of assistantships under competent men. The ability of the senior surgeon to impart his knowledge and skill to the assistant and the clinical opportunities provided are major factors in the end result. Postgraduate education is planned to help the practitioner keep abreast of new developments.

Announcement of the membership, aims and purpose of the American Board of Plastic Surgery, Inc., was made in *The Journal of the American Medical Association.*[18] The aim of the organization "is to encourage the study, improve the practice, and advance the cause of plastic surgery, and to grant and to issue certificates of recognition of special knowledge in plastic surgery to surgeons meeting the requirements." The qualifications required are outlined, and an examination in theory and practice of plastic surgery is prerequisite to certification.

TRANSPLANTATION AND IMPLANTATION OF TISSUE: SKIN GRAFTS, ETC.

According to Brown and McDowell,[19] the satisfactory late function of grafts requires (1) enough skin for free movement, (2) moderate looseness, (3) ability to withstand usual trauma and (4) development of normal sensation. Both thick split grafts and grafts of full thickness seem to grow, or at least to stretch out, with growth of the body areas and to permit normal movement if they have been successful from the start. Repair of large surface defects should be made early if possible. Thick split grafts will work satisfactorily in most cases, and several "crops" of such grafts can be taken from the donor area.

Indications for the use of free grafts and pedicled grafts are outlined by Gerrie.[20] When subcutaneous tissue is also required Wilmoth [21] finds the tubed graft best.

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17. Palmer, A.: Educational Needs in the Development of Plastic Surgery, Eye, Ear, Nose & Throat Monthly **20**:205-208 (Aug.) 1941.

18. American Board of Plastic Surgery, Inc., J. A. M. A. **117**:752-753 (Aug. 30) 1941.

19. Brown, J. B., and McDowell, F.: Persistence of Function of Skin Grafts Through Long Periods of Growth, Surg., Gynec. & Obst. **72**:848-853 (May) 1941.

20. Gerrie, J. W.: The Choice of Skin Grafts in Plastic Surgery, Canad. M. A. J. **44**:9-13 (Jan.) 1941.

21. Wilmoth, C. L.: Tubed Pedicle Graft in Facial Reconstruction: Its Superiority When Subcutaneous Loss Is Present, Am. J. Surg. **53**:300-306 (Aug.) 1941.

In preparing contaminated wounds for skin grafting Veal, Klepser and DeVito [22] found the use of sulfanilamide powder, particularly sulfanilamide (10 per cent)–allantoin (2 per cent) ointment of great value in combating the infection and stimulating the growth of healthy granulations.

The dermatome is an instrument which allows the cutting of precision and uniform skin grafts. Padgett [23] finds that for granulating wounds grafts up to 0.012 inch (0.3 mm.) in thickness will "take" successfully. On aseptic bases, such as those made for release of scars, grafts up to three-fourths the thickness of the skin may be applied with success. Such grafts allow excellent functional use, are pliable and heal with minimal scarring and contracture.

An excellent monograph on the technic of skin grafting is published by Koch.[24] The management of grafts of intermediate thickness and whole skin thickness, pedicled flaps and combined grafts as related to surgery of the hand is described in detail. The basic principles of his masterly technic can be transferred to the management of grafts anywhere in the body.

Implantation of many and large skin grafts in a debilitated patient may require much more time and reserve than the patient can safely afford. Ellis and Von Wedel [25] find that instead of sewing the graft in, collodion applied to the cutaneous surface of the graft, and to a dried bed, will afford excellent fixation and save considerable time.

Barber [26] notes that the injection of minute particles of dermal tissue beneath the surface of a granulating wound appears to stimulate the healing of raw indolent wounds.

In the treatment of pitted scars, pigmentation and some facial blemishes, Eller and Wolff [27] report that skin peeling artificially produced by physical or chemical agents may secure beneficial results. The exfoliation of skin can be controlled to include only the corneous layer, or more if desired.

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22. Veal, J. R.; Klepser, R. G., and DeVito, M. P.: The Preparation of Superficial Wounds for Skin Grafting by the Local Use of Sulfanilamide and Sulfanilamide-Allantoin Ointment, Am. J. Surg. **54**:716-720 (Dec.) 1941.

23. Padgett, E. C.: Skin Grafting and the "Three-Quarter" Thickness Skin Graft for Prevention and Correction of Cicatricial Formation, Ann. Surg. **113**: 1034-1048 (June) 1941.

24. Koch, S. L.: The Transplantation of Skin and Subcutaneous Tissue to the Hand, Surg., Gynec. & Obst. **72**:157-177 (Feb.) 1941.

25. Ellis, S. S., and Von Wedel, C.: Collodion as a Dressing for Skin Grafting of Granulating Wounds, J. Oklahoma M. A. **34**:103-105 (March) 1941.

26. Barber, C. G.: Skin Transplantation by Injection: Its Effect in Healing of Granulating Wounds, Arch. Surg. **43**:21-31 (July) 1941.

27. Eller, J. J., and Wolff, S.: Skin Peeling and Scarification, J. A. M. A. **116**:934-938 (March 8) 1941.

The treatment of various conditions by irradiation may cause such damage to the overlying or the neighboring skin that skin grafting is indicated. The late changes in an irradiated field comprise telangiectasis, followed by hyperkeratoses, alopecia, fissures, fibrosis and later loss of the subcutaneous fat, marked endarteritis of the smaller vessels and degenerative changes in the fascia. The most serious of all complications is the development of carcinoma in the skin of the treated area. There is an incubation period between the use of roentgen rays and the development of cancer of from three to eighteen years. The surgical management, according to Daland,[28] consists of broad excision of the involved skin, followed by either Thiersch grafting or the use of a pedicle flap. Kimball [29] divides irradiation injuries into two types: (1) lesions resulting from a massive dose, with subcutaneous necrosis and pain, best treated by excision and postponement of repair until healthy granulations develop and (2) chronic, long-standing lesions, excision and repair of which can usually be carried out in one stage.

Other conditions requiring similar excision and skin grafting are extensive moles of the face, as reported by Figi.[30]

May [31] lists the three most essential features in achieving a successful transplantation of skin as (1) avoidance of infection, (2) avoidance of hematoma between the graft and the host and (3) complete immobilization of the transplanted area. For transplantation of fat tissue the graft should be two thirds larger than that required to counteract degeneration and shrinkage.

In regard to the transplantation of cartilage, when the graft is taken from the patient himself, it remains alive and forms an organic union with the host tissue. When the graft is taken from another person, it dies, the nuclei disappear, the transplant becomes encapsulated like a foreign body and is apt gradually to become absorbed. The same is true of cartilage preserved in alcohol (Peer) or refrigerated cartilage (O'Connor and Pierce).

Experiments by Young [32] on the transplantation of autogenous cartilage from the rib in dogs, with and without perichondrium, showed that the cartilage remains viable and maintains its size and weight.

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28. Daland, E. M.: Radiation Damage to Tissue and Its Repair, Surg., Gynec. & Obst. **72**:372-383 (Feb.) 1941.

29. Kimball, G. H.: The Surgical Treatment of Radiation Damage to Tissue, J. Oklahoma M. A. **34**:285-289 (July) 1941.

30. Figi, F. A.: Extensive Mole of the Face and Scalp; Excision and Full Thickness Skin Graft, Proc. Staff Meet., Mayo Clin. **16**:280-282 (April 30) 1941.

31. May, H.: Transplantation and Regeneration of Tissue, Pennsylvania M. J. **45**:130-135 (Nov.) 1941.

32. Young, F.: Autogenous Cartilage Grafts: An Experimental Study, Surgery **10**:7-21 (July) 1941.

Dupertuis,[33] experimenting with young cartilage in rabbits, observed that growth of the cartilage occurred. Straith and Slaughter [34] state that in man autogenous cartilage grafts remain as living tissue and neither increase nor decrease. Homografts in human beings may become slightly absorbed, but if the graft is replaced by fibrous tissue, and therefore still maintains its contours, it may constitute a desirable filling substance, even though the original cartilage itself is lost. In 81 of 100 consecutive cases in which homografts were used, recovery was uneventful. In 19 there was some reaction such as redness. Of the 19, the cartilage was retained in all but 4, in which it was lost entirely, and 2, in which it was lost in part. Thus, 94 per cent of the grafts were classed as successful. On 7 of the grafts a second operation was performed to remove or trim the cartilage when curving or migration had taken place.

Iglauer [35] reports similar good results with the use of refrigerated homografts which were kept in a mixture of 1 part aqueous merthiolate (1:1,000) and 4 parts physiologic solution of sodium chloride.

Peer,[36] in investigating the possibilities of using cartilage removed from the septum during a submucous resection as a source of material for a graft, finds that it survives as a living autograft, just as does cartilage which has been removed from a rib.

Mowlem [37] objects to the use of cartilage implants because such a high percentage show curling or migration and require secondary operation. The iliac bone offers material to be used as a graft which survives transplantation successfully and obviates the objections to cartilage. It appears unnecessary to retain the periosteum or to put the bone in contact with the nasal bone for successful results. Sheehan [38] has also used iliac bone successfully in repair of facial and cranial injuries and recommends it highly.

Malbec [39] reports the use of duralumin as an implant in cases of nasal deformities which is tolerated by the tissues.

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33. Dupertuis, S. M.: Actual Growth of Young Cartilage in Rabbits: Experimental Studies, Arch. Surg. **43**:32-62 (July) 1941.

34. Straith, C. L., and Slaughter, W. B.: Grafts of Preserved Cartilage in Restorations of Facial Contour, J. A. M. A. **116**:2008-2013 (May 3) 1941.

35. Iglauer, S.: Use of Preserved Human Cartilage in Reconstructive Facial Surgery, Ann. Otol., Rhin. & Laryng. **50**:1072-1078 (Dec.) 1941.

36. Peer, L. A: Fate of Autogenous Septal Cartilage After Transplantation in Human Tissue, Arch Otolaryng. **34**:696-709 (Oct.) 1941.

37. Mowlem, R.: Bone and Cartilage Transplants: Their Use and Behavior, Brit. J. Surg. **29**:182-193 (Oct.) 1941.

38. Sheehan, J. E.: The Use of Iliac Bone in Facial and Cranial Repair, Am. J. Surg. **52**:55-61 (April) 1941.

39. Malbec, E. F.: El duraluminium en cirurgía plástica, Semana méd. **1**:1016-1019 (May 1) 1941.

PLASTIC SURGERY OF THE NOSE

According to King,[40] in all nasal injuries sustained in childhood a thorough septal examination should be made and correction of any displacement carried out early. External deformity at such a time is usually not evident or is masked by external swelling, but will become manifest with time and increase with age if it is not detected at the time of the accident and corrected.

Metzenbaum [41] classifies as a clinical entity the type of nasal injury in which the blow is severe and directly over the nasal bridge, producing the following changes:

1. A depressed locked fracture of the two nasal bones occurs at about their upper third. 2. The two nasal bones no longer form a V, or a gabled roof, to the bridge of the nose but are driven in between, flattened and depressed to the level of the upper borders of the nasal process of the maxillary bones. 3. This flattening and depression of the nasal bones between the upper borders of the walls of the nasal processes of the maxillary bones increases the width at this plane, and a stress is transmitted within the walls of the nasal processes of the maxillary bones to the nasal base, resulting in a divergent or outward spreading fracture of both outer nasal walls along the base lines of both nasal processes of the maxillary bones.

His treatment consists in exerting pressure with the thumbs against the two divergent lateral nasal walls until they are forced toward each other into their normal anatomic positions. This procedure narrows the nasal base, and at the same time it causes the upper borders of the walls of the fractured nasal processes of the maxillary bones to spread slightly farther apart, so that the depressed locked lower ends of the nasal bones can be unlocked more readily and elevated without splintering. After molding, an external splint is applied to prevent the formation of hematoma.

In the treatment of the extremely deviated nose which is the result of previous trauma and has healed in that position, it is often necessary to remove a small wedge of osseous tissue on the broad side of the nose before repositioning in the midline can be accomplished. Sparer [42] has simplified the removal of such a wedge by inserting a hemostat onto the bared bone and cutting above and below it for removal of a wedge.

Indications for nasal plastic surgery, according to Cohen,[43] are both physiologic and psychologic. Surgery in this field has reached such a

40. King, E. D.: Plastic Surgery of the Nose and Face, West Virginia M. J. **37**:293-297 (July) 1941.

41. Metzenbaum, M. F.: Recent Fracture of the Nasal Base Lines of Both Outer Nasal Walls, with Divergent Displacement, Arch. Otolaryng. **34**:723-735 (Oct.) 1941.

42. Sparer, W.: Nasal Plastic Operation to Cure Large Hump with Deviation of Nasal Axis: Modified Technic, M. Rec. **153**:92-94 (Feb. 5) 1941.

43. Cohen, S.: Plastic Surgery of the Nose, Laryngoscope **51**:363-377 (April) 1941.

development that the ultimate aim today is to secure as nearly a perfect result as possible. Complications due to poor technic or poor judgment are frequent, while those due to infection are infrequent and rarely serious. In the evaluation of the patient the age factor is of importance. Correction of septal displacements noted early in life can be easily made by replacement methods and will save much subsequent deformity. On the other hand, narrowing or raising the nose is best approached surgically when complete growth has taken place. In the management of specific deformities, such as the traumatic saddle nose, correction is often possible by utilizing the bony and cartilaginous tissue available within the nose. The method of correcting a deflected hump nose which Cohen uses consists of removing the bony hump at an angle so that the remaining side walls are then of equal length and can be fractured over to the midline.

A rare deformity due to an intranasal granuloma of the sporothrix type is reported by Ortmayer and Humphreys.[44]

In the treatment of cleft lip deformities, the more difficult part of the operation is to secure good correction of the associated nasal deformity. Secondary operation for nasal deformity persisting after an operation for cleft lip is often necessary later in life. According to Cinelli,[45] the nasal deformity is characteristic of this type of malformation. The tip of the nose is flattened; drooping of the alar rim gives the nares a flaring effect, making the base of the nares unduly wide. The septum is usually deviated to the normal side. Surgical correction consists chiefly of remolding the alar cartilages and narrowing the nasal floor. According to Brown and McDowell,[46] correction of the nose in cases of cleft lip may require intranasal osteoplastic reduction in the size of the nose with reconstruction of the lateral cartilages. Secondary repair of the lip may include excision of scars, adjustment of levels and transfer of vermilion border flaps from the lower lip. In cases of double cleft lip, advancement of the columella to give prominence to the nasal tip is often indicated, as well as a dental prosthesis to hold the lip out in full contour.

For some reason a prosthesis to replace part or all of the nose may be necessary for temporary or permanent use. The prominence of the

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44. Ortmayer, M., and Humphreys, E. M.: Intranasal Granuloma of the Sporothrix Type Producing Marked Nasal Deformity, Ann. Surg. **113**:118-123 (Jan.) 1941.

45. Cinelli, A. A.: Secondary Nasal Deformities Following Correction of Cleft Lip, Laryngoscope **51**:1053-1058 (Nov.) 1941.

46. Brown, J. B., and McDowell, F.: Secondary Repair of Cleft Lips and Their Nasal Deformities, Ann. Surg. **114**:101-117 (July) 1941.

nose requires exacting attributes in the prosthesis to be cosmetically acceptable. Bulbulian [47] finds that the most satisfactory material for such a prosthesis is a latex compound.

PLASTIC SURGERY OF THE FACE

On investigation of the decisive factors in the production of fine linear scars in surgical procedures, Cox [48] concludes that it is not any particular method of wound suture which is important, but rather the relation which the direction of the incision bears to the cleavage line of the skin. Extrinsic factors influence the originating and molding of the line pattern, but the maintenance is purely intrinsic.

In discussing the treatment of acute traumas of the skin and subcutaneous tissues, Bancroft [49] states that many of the failures in the management of such conditions are due to lack of appreciation of the pathologic process occurring in the subcutaneous fat and connective tissue. The success or failure of the general treatment of contused wounds of the skin depends largely on tissue tension. A hematoma should be aspirated, and when severe relaxation incisions should be performed. One might summarize the problem of primary or secondary closure of lacerated wounds as follows: Primary closure may be attempted if the patient is healthy, the skin is clean, the instrument causing damage is relatively clean and sharp and the patient has been seen well under six hours after the injury. In regard to avulsed wounds, Farmer's method of excision of the avulsed piece, with removal of all fat and resuture into the wound as a free graft, is advocated.

The treatment of war wounds is a problem of major importance. Ivy and Stout,[50] in discussing the emergency treatment of war wounds of the face and jaws, report that the points demanding special attention in the combat area are: (1) arrest of hemorrhage, (2) provision of an adequate airway, (3) temporary reduction and fixation of bone fragments and (4) provision for safe transportation.

The main object in the primary care of injuries of the face and jaws, according to Kazanjian,[51] is to reestablish function of the various tissues

47. Bulbulian, A. H.: Reconstruction of Facial Defects with Latex Compound: Results Obtained Recently, Proc. Staff Meet., Mayo Clin. **16**:313-318 (May 14) 1941.

48. Cox, H. T.: The Cleavage Lines of the Skin, Brit. J. Surg. **29**:234-241 (Oct.) 1941.

49. Bancroft, F. W.: Treatment of Traumas of Skin and Subcutaneous Tissues, Surg., Gynec. & Obst. **72**:318-327 (Feb.) 1941.

50. Ivy, R. H., and Stout, R. A.: Emergency Treatment of War Injuries of the Face and Jaws, Ann. Surg. **113**:1001-1006 (June) 1941.

51. Kazanjian, V. H.: Primary Care of Injuries of Face and Jaws, Surg., Gynec. & Obst. **72**:431-436 (Feb.) 1941; Primary Care of Injuries of Face and Jaws, Am. J. Orthodontics **27**:448-458 (Aug.) 1941.

of the face and jaws and to eliminate, or at least minimize, the visible deformity. The ideal time for suturing the soft tissues of the face is at least within five to six hours after the accident. Not all wounds of the face are cleancut injuries, ready for suturing. There is a great temptation to repair the lacerated and edematous tissues as soon as possible; yet one must remember that suturing of even mildly infected wounds never gives a good cosmetic result and complications may be caused from too early closure. Late primary suture consists of (1) thorough mechanical cleansing of the wound, (2) application of frequent hot fomentations of boric acid and (3) maintenance of free drainage of the wound. Experience has shown that even badly infected and bruised facial wounds can be made ready for suturing in from two to ten days by this method. In the treatment of fractures of the jaw early and adequate immobilization is the first principle. The fundamental aim is to bring the existing teeth into their normal alinement. This holds true even if there is a definite gap between the two fragments of the mandibular bone. Management of injuries about the eyelids is particularly important to prevent notches, adhesions and ectropion. Spaeth [52] recommends that the suturing of all through and through tears be made meticulously in layers. To prevent notching, he uses Weiner's method of slightly cutting the lips of the wound crescentically. If possible, vertical lacerations should be inclined away from the perpendicular by using triangular resections in order to prevent notching and to improve the scar line. Other valuable papers on the treatment of maxillofacial injuries are contributed by Owens and Vincent,[53] Ivory [54] and Padgett.[55]

The makeup of the team handling maxillofacial injuries is important. Blair,[56] reporting on experiences in World War I, states that the average dental surgeon has special knowledge of the treatment of fractures of the jaw. The well trained surgeon is skilled in the care of the tissues of the whole body, and by pairing these specialists—the dentist and the physician—into individual working units it is possible to put the care of injuries of the face and mouth on a par with other standardized

52. Spaeth, E. B.: The Immediate and Late Care of Injuries About the Orbit, Surg., Gynec. & Obst. **72**:453-465 (Feb.) 1941.

53. Owens, N., and Vincent, R. W.: Management of Facial Injuries. New Orleans M. & S. J. **94**:221-232 (Nov.) 1941.

54. Ivory, J. J.: Maxillo-Facial Injuries: Important Considerations. Indust. Med. **10**:52-54 (Feb.) 1941.

55. Padgett, E. C.: Severe Injuries of the Face and Jaws, Am. J. Surg. **51**: 828-846 (March) 1941.

56. Blair, V. P.: The Role of the Plastic Surgeon in the Care of War Injuries, Ann. Surg. **113**:697-704 (May) 1941.

procedures. Kazanjian [57] also agrees as to relation of the dentist and the surgeon in the treatment of deformities of the face and jaws.

Wartime experience with fractures of the facial bones and jaws is reported on in detail by McIndoe.[58] In commenting on fractures of the middle third of the face, he states there is a widely held opinion that crush fractures in this area are best left alone in the early stages and should be sent for special treatment later. It cannot be too strongly emphasized that this attitude is entirely wrong. To obtain good results, early diagnosis, immediate disimpaction and immobilization are essential.

In reviewing 119 cases of wartime fracture of the jaw McIndoe [59] notes that the causes, distribution and variety differ greatly from those seen in civilian practice and that their management presents new problems. In this series, bombs, mine explosions and penetrating missiles accounted for 45 per cent of injuries; the collapse of buildings produced 13 per cent, while head-on crashes of various vehicles caused 36 per cent. Immediate treatment consists of cleansing the wound, suturing with drainage in cases of compound fracture of the jaw and immediate fixation of the jaw with intermaxillary wires. Later, cast metal splints are highly satisfactory. Extensive loss of bone in the mandible, requiring bone grafting, occurred in 10 per cent of the cases.

Other methods of fixation of the fragments in cases of fracture of the jaw are described by Fish.[60] He utilizes bands placed on some of the teeth as fixation points and attaches an arch wire bucally to hold the parts together. Cap splints have proved to be valuable in the experience of the British, and a masterful illustrated exposition of this method of management is published by McLeod and Shepherd.[61]

The use of intermaxillary elastics in the management of fracture of the jaw is often feasible and allows opening of the jaws and some motion. Strock [62] describes a method he has found serviceable.

For fracture of the maxilla and accompanying fractures of the facial bones, extramaxillary traction and immobilization are often

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57. Kazanjian, V. H.: Interrelation of Dentistry and Surgery in the Treatment of Deformities of the Face and Jaws, Am. J. Orthodontics **27**:10-31 (Jan.) 1941.

58. McIndoe, A. H.: Diagnosis and Treatment of Injuries of the Middle Third of the Face, Brit. Dent. J. **51**:235-245 (Oct.) 1941.

59. McIndoe, A. H.: Surgical and Dental Treatment of Fractures of the Upper and Lower Jaws in War Time, Proc. Roy. Soc. Med. **34**:267-288 (March) 1941.

60. Fish, E. W.: Some Aspects of Jaw Surgery, Brit. Dent. J. **70**:253-264 (April) 1941.

61. McLeod, A. C. R., and Shepherd, P. R.: Cap Splints, Brit. Dent. J. **71**:268-277 (Oct. 15) 1941.

62. Strock, M. S.: Fractures of the Mandible, Surg., Gynec. & Obst. **72**:1047-1051 (June) 1941.

necessary. Bisnoff [63] has constructed a universal head cap to give such support wherever needed.

Fracture of the zygomatic bone is not uncommon in wartime and in present day traffic injuries and is often undiagnosed because of overlying swelling and ecchymosis. In cases of severe injury in which there is also comminution of the anterior and posterior sinus walls, the fragments of the zygomatic bone slip back into the depressed position when the elevating force is removed. In such cases Baxter [64] uses a metal splint wired to the teeth as a basic support for a prop which extends up through the alveolobuccal sulcus to engage and hold the fractured zygoma in position until a supporting callus is formed.

Reconstructions about the face are often necessary in the treatment of cancer of the face or after the eradication of malignant growths by roentgen irradiation and radium therapy. New [65] presents a surgical clinic on the treatment of such conditions. A clinic on congenital and acquired nonmalignant lesions of the face is presented by Jenkins.[66]

Deformities of the lips often occur in the management of congenital cleft lip, particularly double cleft lips. Cannon [67] recommends using the prolabium for the central portion of the lip in primary repair, so that the wound can be closed with minimal tension and yet have the lip and vermilion border resemble the normal. When the child is 10 to 12 years old, the prolabium can be excised and replaced by a split vermilion-bordered lip flap from the lower lip.

For the loss of more than two thirds of the lower lip in cases of resection for carcinoma or for defects resulting from irradiation, repair is usually carried out by flaps taken from distant points, involving much time and several operations. Transplantation of local tissues can be carried out by May's [68] operation in one stage. The author uses a heart-shaped excision of the tumor. The incisions on either side meet each other at a point behind the central part of the mandible. This point becomes the point of rotation around which two square flaps are turned from the immediate neighborhood.

Some facial defects are best handled by a suitable prosthesis. To facilitate the taking of an agar compound impression of the defect,

63. Bisnoff, H. L.: Fractures of the Jaws and Facial Bones, Dent. Outlook **28**:343-347 (Aug.) 1941.

64. Baxter, H.: A New Method of Treatment of Depressed Fractures of the Zygomatic Bone, Canad. M. A. J. **44**:5-9 (Jan.) 1941.

65. New, G. B.: Treatment of Cancer of the Face Including Reconstructive Surgery, S. Clin. North America **21**:969-978 (Aug.) 1941.

66. Jenkins, H. P.: Plastic Surgery of the Head and Face, S. Clin. North America **21**:37-53 (Feb.) 1941.

67. Cannon, B.: The Split Vermilion Bordered Lip Flap, Surg., Gynec. & Obst. **73**:95-98 (July) 1941.

68. May, H.: One Stage Operation for Closure of Large Defects of Lower Lip and Chin, Surg., Gynec. & Obst. **73**:236-239 (Aug.) 1941.

Kaufman [69] devised a matrix made of brass tubing through which water can be run in order to hasten the setting of the impression material. Bulbulian's [70] researches establish the superior qualities of a latex compound for the average external prosthesis. For the larger restorations, involving body cavities as well, acrylic resin, as used by Munson and Heron,[71] is preferable. One interested in the surgical rehabilitation of patients with deformities of the face and mouth subsequent to the treatment of cancer of these regions can ill afford to miss the superb book by Blair, Moore and Byars.[72]

PLASTIC SURGERY OF THE EAR

Defects of the ear are congenital or acquired. In congenital defects the auricle may be partially or wholly missing. Acquired defects are usually due to (1) trauma, (2) infection or (3) a malignant growth. Cox [73] repairs partial losses by means of a folded flap taken from the postauricular area. Total reconstruction is carried out by the method described by Pierce. Newman [74] also advocates this method.

According to Greeley,[75] the chief stumbling block in rebuilding a new ear lies in the problem of furnishing an adequate support to the overlying skin. Prior to the more recent procedure the best attempt to supply a supporting framework consisted in the utilization of costal cartilage. A distinct advance has been made by using cartilage from the ear of another person, usually the mother. The sacrifice of maternal cartilage is not as mutilating to the donor as one might think.

A new operation for repair of postoperative mastoid fistula is reported by Lillie and Pastore.[76] The technic is as follows: 1. Initial excision of the scarred edges and liberal freeing of the tissues laterally are carried out. 2. The edges of the wound are split into a V, so that an overlapping subcutaneous closure can be effected. 3. Stay sutures tied over rubber tubing are used to evert the edges and produce even pressure.

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69. Kaufman, J. L.: Facial Masks: A New Technic Using a Newly Devised Matrix, J. Am. Dent. A. **28**:64-66 (Jan.) 1941.

70. Bulbulian, A. H.: Prosthetic Reconstruction of Nose and Ear with a Latex Compound, J. A. M. A. **116**:1504-1508 (April 5) 1941.

71. Munson, F. T., and Heron, D. F.: Facial Reconstruction with Acrylic Resin, Am. J. Surg. **53**:291-296 (Aug.) 1941.

72. Blair, V. P.; Moore, S., and Byars, L. T.: Cancer of the Face and Mouth, St. Louis, C. V. Mosby Company, 1941.

73. Cox, G. H.: Surgery of the Auricle, Including Total Reconstruction and Protuberant Ears, Laryngoscope **57**:791-797 (Aug.) 1941.

74. Newman, J.: Reconstruction of the Ear, Surg., Gynec. & Obst. **73**:234-235 (Aug.) 1941.

75. Greeley, P. W.: Reconstructive Otoplasty, Surgery **10**:457-462 (Sept.) 1941.

76. Lillie, H. I., and Pastore, P. N.: Repair of Postoperative Mastoid Fistula: Report of a Method, Arch. Otolaryng. **34**:687-695 (Oct.) 1941.

Abstracts from Current Literature

Ear

DISCUSSION ON FUNCTIONAL DEAFNESS. Proc. Roy. Soc. Med. **33**:463 (June) 1940.

Sir Arthur Hurst discussed the etiology, diagnosis and treatment of the hysterical form of deafness, having had the opportunity of observing many patients with this condition during the last war. He contended that when a man is temporarily deafened by a loud noise or by some recoverable disease he finds that he is unable to hear, however much he tries, and consequently after a time he gives up trying. When the cause of the deafness then disappears he has become so convinced that he cannot hear that he makes no further attempt to listen. He is then suffering from hysterical deafness. Hurst's experience led him to the following conclusions: 1. Bilateral deafness following the explosion of a powerful shell is generally hysterical. 2. Mutism and tremor are commonly associated. 3. The supposed association of hysterical anesthesia of the external ear is a complete fallacy. 4. When the deafness is not absolute, a tuning fork can still be heard by bone conduction. Many patients, however, show total deafness. 5. In hysterical deafness the deafness persists during sleep. 6. In deep hypnosis no response to commands can be obtained and no auditory-motor reflex is produced. 7. The majority of patients with hysterical deafness show no change in the timbre or intonation of the voice. 8. The only test on which almost complete reliance can be placed in the differentiation of absolute hysterical deafness from absolute organic deafness is the presence of normal vestibular reactions to rotation and caloric tests in the former and their loss in the latter.

In discussing the treatment of hysterical deafness, Hurst stated that when deafness is associated with mutism the hearing returns when speech is restored. The mutism is corrected by simple explanation and persuasion. Uncomplicated hysterical deafness is more difficult to treat. A large majority of the patients respond to explanation, persuasion and reeducation.

Dr. Emanuel Miller discussed the subject chiefly from the standpoint of various factors involved in the causation of functional deafness and psychologic factors concerned in its successful treatment.

CAMPBELL, Philadelphia.

MENGONI'S TEST FOR SIMULATION OF UNILATERAL DEAFNESS. S. MENGONI, Arch. ital. di otol. **52**:588 (Nov.) 1940.

Mengoni declares that his test is conclusive in 99 per cent of cases. The test is applicable only to persons who have good functional hearing in one ear. With the "deaf" ear tightly closed with the finger, the examiner pronounces some words in the barest whisper with his lips almost touching the auricle. Naturally, the patient claims not to hear anything. The "sound" ear is closed, and the same procedure is carried out with the other ear. But while the test is in progress the finger is unexpectedly removed from the sound ear; the patient then, believing he hears with the unstopped ear, will repeat the test words. If he still declares he is unable to hear the words, the simulation is revealed, for even if there were deafness in one ear, he could still hear the words in the sound ear.

DENNIS, San Diego, Calif.

STATISTICS ON ETIOLOGIC EXAMINATIONS CONDUCTED AT THE COUNTY SCHOOL FOR THE DEAF IN MUNICH. E. EMMERIG, Monatschr. f. Ohrenh. **74**:425 (Sept.) 1940.

With the law purporting to prevent inheritance of diseases or disease tendencies (*Erbkranke*) in mind, the students of a school for deaf children were

examined and the statistical results evaluated. Of 237 children examined, deafness was inherited (the author says "perhaps") in 30 per cent, congenitally acquired (without apparent reason) in 27 per cent and acquired in 35.4 per cent and in 7.6 per cent it was of uncertain origin. This is interpreted as indicating a decrease in acquired deaf mutism since the beginning of the century. On the basis of a study of ascendants in cases of inherited deafness, indirect transmission seems to prevail. An interesting sidelight is revealed, namely, that in families with deaf mutism the average number of children is twice that in normal families (5 : 2.35) and that twin births are more frequent among these families. Only 18 per cent of the children were totally deaf, while 43.3 per cent had sufficient hearing left to enable them to learn by hearing rather than by lip reading or other methods. Separation of these two groups for teaching purposes seems advisable. Langenbeck's rule of symmetric defects as an indication of inherited deafness should not be overestimated, inasmuch as the sign was not present in 21 per cent of children with inherited deafness who were examined. Several tables support the author's conclusions. In cases of the acquired form of deafness the etiologic factors given, in order of importance, are cerebral disease, disease of the ear, measles, trauma, forceps delivery, syphilis, whooping cough, diphtheria and grip. Many of the patients had sisters or brothers who were hard of hearing. In very few cases was deafness due to consanguinity.

LEDERER, Chicago.

AUDIOMETRIC RECHECK OF RESULTS OF THE "HORMONAL" TREATMENT OF ZAJIČEK. H. MELLER and G. E. ARNOLD, Monatschr. f. Ohrenh. **74**:444 (Sept.) 1940.

In a series of 25 cases the effect of the use of ointments and serums containing extracts of calf testicles and ovaries on organic deafness, as advocated by Dr. Zajiček, was checked with the aid of voice tests, tuning forks and an audiometer (6A Western Electric). The ointment was applied with "force" to the neck and shoulder and also the auricle of the affected ear. No objective improvement could be observed, and in the few cases in which subjective impressions of improved hearing were obtained these results were not permanent. The author comments on the psychologic aspects of such examinations, the effect of studies on unintelligent patients and the influence of fatigue and other factors which might contribute to a margin of error in the determinations.

LEDERER, Chicago.

INFLUENCE OF CHANGES IN TEMPERATURE ON HEARING. G. JANKOFF, Monatschr. f. Ohrenh. **75**:1 (Jan.) 1941.

Occupational deafness caused by noise or percussion is well recognized, but not so the disturbance of hearing caused by abrupt, extreme changes in temperature. Temkin stated that this influence is achieved by way of the eustachian tubes. Jankoff contends that these changes affect the cochlea directly. He discusses at length the anatomy of the ear and concludes that changes in the blood supply influence the response of Corti's organ. The blood supply depends on the width of the supplying vessels, changes being followed by anemia or hyperemia, stasis and edema. The influence of changes in temperature on vessels is well known and may even cause vasoparalysis, with subsequent minute bleeding. To prove this contention, 310 railway engineers and stokers were examined over a period of years and their working habits and conditions studied. A total of 22 cases are cited in which definite loss of hearing occurred over this period. In all of them the middle ear was normal and a positive Rinne reaction and a shortened Schwabach time were demonstrated. All ranges of the cochlea were proved to be involved (no audiometer was employed), the right ear being worse than the left. Investigation of their working conditions showed that the right ear was more exposed to changes in temperature, as the head was usually protruded partly out of the right side of the engine caboose. The temperature of the caboose and that outside was shown to differ from 20 to 45 C. A study of the influence of percussion and

noise proved that these factors are less disturbing in the engine than in any other car. Brakemen, examined for comparison, evidenced only equal loss of hearing in the two ears. A series of guinea pigs were treated with applications to the head, the temperature of which varied from hot to freezing cold through different periods. Histologic examination of the cochlea of these animals showed marked signs of degeneration, involving nearly all constituents, especially the nerve elements. The changes are illustrated in five photomicrographs. Ten cases are presented in detail.

LEDERER, Chicago.

A CLOUD WITH A SILVER LINING IN OTOSCLEROSIS? M. BAER, Monatschr. f. Ohrenh. **75**:22 (Jan.) 1941.

After a general discussion of the properties of vitamin E, the cases of 10 patients treated with vitamin E are reported. Five of the patients with otosclerosis did not improve at all; 2 improved slightly in their conversational hearing, and 3 had a decided increase in conversational hearing and a subjective decrease of tinnitus. Treatment was continued over at least three months, and a daily average of 60 to 90 mg. of alpha tocopherol was given by mouth. This was later reduced to from 20 to 30 mg. No ill effects were observed, and the treatment is recommended especially for young persons and for those who present no far advanced signs of otosclerosis. Incidentally, the author states that "otosclerosis is a relatively rare disease."

LEDERER, Chicago.

PATHOLOGIC ASPECTS OF MASTOIDITIS. T. NISHIHATA, Monatschr. f. Ohrenh. **75**:31 (Jan.) 1941.

The author "rediscovers" mastoiditis. In 5 patients with acute otitis media who were subjected to operation on the second to the fifth day of the attacks he found that all the mastoid cells had signs of inflammation and suppuration. In 9 patients on whom operation was performed eleven to forty-three days after the onset of the otitis only partial involvement of the mastoid cells was present (but no mention is made as to whether surgical intervention was indicated). The author believes that the extension of the inflammatory process follows Stenger's route of invasion. The antrum may be free of involvement while cells of the posterior wall of the auditory canal and the *Schwellenzellen* may show pathologic changes, so that direct extension from the tympanic cavity may be suspected. Histologic studies are discussed (but not shown) to support the contention that there exists a latent stage between otitis media and mastoiditis and that a special nomenclature should be applied, such as the "latent stage of mastoiditis" or the "mastoid cells form of otitis media." Portmann's empyema of the mastoid may be an equivalent, but while Portmann described condensing osteitis, followed later by a ramifying osteitis throughout the mastoid process, the author believes that much greater changes are present within the cells of the route of extension. Therefore, the deeper cells are inflamed much earlier than was previously believed, and destruction of bone is present earlier than in the surrounding cells. These cells, meanwhile, evidence new bone formation. The observations are essentially the same as Portmann's, but the explanation differs in the main in the fact that the deep cells are affected earlier.

LEDERER, Chicago.

TREATMENT OF PROGRESSIVE DEAFNESS BY MEANS OF "HORMONE" THERAPY ACCORDING TO THE ROUTINE OF ZAJIČEK. R. PERWITZSCHKY and J. NASEMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:418 (June) 1940.

Perwitzschky and Nasemann studied the effect of "hormone" therapy of diseases of the internal ear in a group of 33 patients. The patients showed progressive deafness, presbycusis and other evidences of a pathologic condition of the internal ear. The authors followed the technic of Zajiček, including the preparation of the solution to the minutest detail. They found that there was absolutely no

improvement from this method of treatment. Anterior pituitary extract, gonadotropin, estrogen and androgen were also used. The authors concluded that all of these preparations are valueless as an influence on progressive deafness.

PERSKY, Philadelphia.

DISTRIBUTION OF OTOSCLEROSIS AND EXPERIMENTAL STUDIES CONCERNING ITS SURGICAL TREATMENT. H. ENGSTRÖM, Acta otolaryng., 1940, supp. 43.

Pathology of Otosclerosis.—One hundred and forty-five temporal bones selected at random from 100 persons, of whom 50 were males and 50 females, were studied as to the presence of otosclerosis. The following observations were made:

1. There were otosclerotic changes in 12 cases—in 8, of the spongy type, and in the remaining 4, merely of the "blue mantle" type.

2. In 6 cases in which both temporal bones were examined the otosclerotic changes were bilateral and fairly symmetric.

3. In all 12 cases the otosclerosis occurred in persons over 40 years of age. Of these, 8 were males and 4 females. Thirty patients between the ages of 20 and 40 were also examined and no otosclerotic foci were disclosed.

4. The otosclerotic changes represented for the most part early developmental stages of the disease, and in no case was there bony ankylosis of the stapes joint. In 5 cases the changes were found in the typical location, anterior to the oval window.

5. In 2 cases there were circumscribed otosclerotic foci in the labyrinthine capsule which had no connection with any portion of the capsule having to do with sound conduction or perception.

6. Numerous transition stages between the typical spongy otosclerotic changes and the "blue mantle" designate the latter as a developmental phase in the course of otosclerosis.

Experimental Studies on the Surgical Treatment of Otosclerosis.—For this purpose large numbers of monkeys (Macaca rhesus) were used. These experiments showed, first, the normal processes of repair when a fistula is formed in one of the semicircular canals and, second, how this reparative process is influenced by various substances, such as metal, epithelium or radium.

1. If one creates a fistula in a semicircular canal with a gouge, connective tissue closure of the fistula begins after a few days. Bone begins to regenerate after one week. In about twenty-five to thirty days the fistula is completely closed by new-formed connective tissue, osteoid tissue and a true bony callus.

2. If the fistula is produced by a polishing burr, callus formation can be limited to a degree. The author believes this is due to a burning effect of the burr on the bone. If the burr is cooled with carbon dioxide there is no inhibiting effect on callus formation.

3. The size of the fistula has a decided effect on bone-regenerating properties. If a fistula is made which is smaller than one-half the circumference of the canal, one obtains, after healing has taken place, a somewhat smaller spherical opening. If the fistula is wider than half the circumference of the canal, then the entire bony canal fills with new-formed bone and the membranous canal comes to lie in the midst of this new-formed bone as a small tube. It is therefore of prime importance not to make a large fistula.

4. The position of the fistula is of great importance. It should not be placed too close to the ampullar end, where there is danger that the new-formed bone will invade the ampulla and destroy the sensory epithelium and thus prevent a second operation on the same canal.

5. The bone which forms within the territory of the fistula consists of irregularly arranged fibrillae, which later are changed to lamellar bone. This process proceeds very slowly, and only in isolated cases can one speak of true osteoclast resorption.

6. If bone splinters are left behind, these seem to have a pronounced stimulating effect on bone regeneration within the fistula. These bone splinters can remain unchanged within the new-formed bone for as long as four hundred and sixty-five days (the longest period of observation). Therefore, in clinical operations it is highly important to remove all chips or splinters of bone from within the territory of the fistula.

7. If fat, connective tissue, muscle tissue or any other mesodermal tissue is placed against the fistula, it does not in any way interfere with callus formation in the fistula, but rather seems to promote a more rapid bony closure.

8. Paraffin, rubber or the lens tissue of an eye does not seem to interfere with bone regeneration.

9. Experiments with a Thiersch graft of pavement epithelium or with a plastic flap from the membrana tympani do have a distinct slowing effect on callus formation, but in the monkey this was not sufficient to prevent closure of the fistula by bone.

10. The use of thin sheets of metal against the new-formed fistula did act to hinder the bony closure of the fistula to some extent. In this regard copper had the best effect, silver less and gold leaf very little. This limiting effect of the metal seemed to be both chemical and mechanical.

11. The apposition to the new-formed fistula of a stainless steel bullet containing radium seemed to have the greatest effect in limiting new bone formation, but this effect ceased soon after removal of the radium.

This study is an excellent one, with a complete bibliography of the existing literature.

GROVE, Milwaukee.

Pharynx

DISCUSSION ON THE INDICATIONS FOR REMOVAL OF TONSILS AND ADENOIDS IN CHILDREN. Proc. Roy. Soc. Med. **33**:347 (April) 1940.

Sir Lancelot Barrington-Ward stated that a septic tonsil, whether large or small, should always be removed. He expressed the belief that a septic condition of the tonsil can best be determined by a study of the secondary effects, which may be direct or indirect. Among the direct effects he included repeated attacks of tonsillitis, chronic enlargement of the upper deep cervical lymph gland (Wood's tonsillar gland) and otitis media. In discussing the indirect effects he mentioned that rheumatism is less likely to attack persons who have undergone tonsillectomy and that nephritis is often secondary to tonsillitis. He expressed the belief that the least definite of all indications is persistent nasopharyngeal catarrh and the common cold.

J. D. Kershaw, in a survey, had found that persistent and recurrent sore throat, excessive susceptibility to colds and cervical adenitis were constantly relieved by the operation, but investigation of approximately 1,000 children who had had notably enlarged tonsils and had not been operated on showed that tonsillar enlargement per se, even when associated with these symptoms in a moderate degree, showed a tendency to spontaneous cure with increasing age in some 90 per cent of cases.

T. S. Rodgers said he believed there is only one constant indication for tonsillectomy, namely, repeated attacks of tonsillitis from which the patient does not recover completely in the intervals. His indication for removal of adenoids is nasal obstruction which will not yield to conservative treatment. He stated that 200,000 tonsillectomies were estimated to have been done each year in England, with a mortality rate of 85 in 1937.

Josephine Collier discussed the physiology of the tonsils and various operative indications and expressed the belief that in considering tonsillectomy each patient should have a well considered judgment on the relation between the condition of the tonsils and adenoids and the symptoms from which he is suffering.

Musgrave Woodman stressed the importance of the local condition of the tonsils in considering operation.

Hajek said he could not find any proof of the alleged connection between tonsillitis and sinal infection. He stated that in Vienna very few pulmonary complications were observed to follow this operation.

CAMPBELL, Philadelphia.

Larynx

PHYSIOLOGY OF THE VOICE. O. GOEBEL, Monatschr. f. Ohrenh. **74**:263 (June) 1940.

After a discussion of the physical bases for tone production in the human larynx, a study of the tension of the elastic tissue of the cords and larynx as a whole is reported and the importance of the tranversus tension emphasized Examination of fresh specimens revealed that the false cords counteract extreme abduction. The strength of a "posterior" ligament in the cricoarytenoid joint influences the movement of this joint and thereby also the movement of the cords. The position of this joint on the cricoid plate, in the male located in the posterior third of the circumference and in the female in the posterior half, close to the midline, will further influence the amount of possible tension of the vocal muscles and cords.

LEDERER, Chicago.

Nose

THE TREATMENT OF CHRONIC OBSTRUCTIVE RHINITIS WITH SUBMUCOSAL INJECTIONS OF SODIUM PSYLLIATE. E. A. THACKER, Ann. Otol., Rhin. & Laryng. **49**:939 (Dec.) 1940.

The author gives as the indications for this treatment (1) chronic nasal obstruction due to engorged turbinates which have failed to respond to treatment for one month; (2) postnasal discharge, chronic pharyngitis, headache and neuralgic pains in the head and neck in cases in which intumescence of the turbinates exists and is causing pressure on the lateral wall of the nose or septum; (3) intermittent nasal obstruction of long standing, especially when associated with changes in temperature and humidity; (4) long-standing chronic sinusitis with associated turgescence of the turbinates, and (5) good shrinkage of the turbinates with astringents.

Contraindications to the treatment are (1) chronic systemic disease, such as hypertensive or other types of advanced cardiac disease, arteriosclerosis, nephritis and such specific diseases and allergic conditions of the nose as should respond to proper treatment of the primary cause, and (2) anatomic defects of the nasal passages. If surgical correction does not relieve obstruction after several months, injection therapy should be considered; since there is little or no shrinkage of the soft tissues in cases of true hyperplastic rhinitis, patients with this condition will not respond to sclerosing agents.

Technic of Injection.—A 5 per cent solution of sodium psylliate (a 5 per cent solution of the sodium salts of fatty acids of the oil extracted from a seed of the psyllium group) is used. 1. Tampons are saturated with a local anesthetic which does not shrink the tissues (the author uses 2 to 4 per cent butyn sulfate). 2. A spinal puncture needle on a tuberculin syringe is best for injecting the solution into the inferior turbinate, and a long gold needle, which can be bent to fit the need, seems best for the middle turbinate. 3. After removal of the tampons the needle is inserted toward the posterior end of the turbinte, and as it is gradually withdrawn 0.25 to 0.5 cc. of the solution of sodium psylliate is released into the tissues. 4. A cotton pledget is inserted to control bleeding from the puncture. After five minutes this is replaced by cotton saturated with ephedrine or neo-

synephrin hydrochloride. 5. The patient is told to use ephedrine or neosynephrin every two hours for five days. 6. Injection is made on only one side at a sitting, and two or three weeks should elapse between injections.

The author says that there is little pain or discomfort following the injection, and no ulcerations or adhesions are seen. He feels that the treatment has distinct advantages in selected cases. Case reports are given.

M. V. MILLER, Philadelphia.

THE USE OF COLLOIDAL THORIUM DIOXIDE IN ROENTGENOGRAPHY OF THE PARANASAL SINUSES. RUSSELL C. GROVE, Laryngoscope **50**:1165 (Dec.) 1940.

Dissatisfied with poppyseed oil containing 40 per cent iodine, Grove used an aqueous colloidal solution containing 25 per cent thorium dioxide as an opaque medium in roentgenographic examination of the paranasal sinuses of more than 75 allergic patients. The preparation was easily injected into the paranasal sinuses and cast a satisfactory shadow, was not absorbed and was not irritating or toxic.

WOOD, Newark, N. J.

EXAMINATION OF THE SENSE OF SMELL WITH SPECIAL REFERENCE TO TRUE AND SIMULATED ANOSMIAS. L. LÖHNER, Monatschr. f. Ohrenh. **74**:479 (Oct.) 1940.

Only a limited group of odors, highly molecular and with only a slight tendency to evaporate, stimulate solely the olfactory region. The majority of odors, commonly so called, stimulate not only the olfactory fibers but the nerve endings which convey the sense of taste, heat, pain, etc. (*Mischreizduftstoffe*). In a case of true central anosmia a *Mischreizduftstoff* should, therefore, produce some kind of perception sensation, such as stinging or cold, while a pure odor gives no such response. To determine this response a Skramlik setup is used, consisting of a rotating disk on which 50 bottles are placed. They are arranged in such a manner that they can be brought to the patient by means of a nose funnel without his knowledge of which one is being used. An air current is conducted through the bottles with about equal intensity. In the setup 12 pure odors and 30 mixed, irritating odors are used, 5 bottles containing water or air serving as control material. It is advisable to use an optimum temperature of 18 C., inasmuch as certain pure odors change their character to mixed forms at a higher temperature. Some of the testing substances create even more than two sensual impressions. According to their stimulation value, the author chose 10 *Mischreizduftstoffe* for the combination of smell and taste (5 for the sour quality and 5 for the sweet); 10 were used to create a cold or heat compression and 10 for stinging pain, through their tactile impression. For practical use, 16 test bottles are considered sufficient, and tables included in the article give complete details of the materials employed. Examination of the patient should be repeated several times, without causing too much fatigue. Results may be expected as follows: (1) normal response; (2) reaction to strong irritation in both categories and no response to low grade stimulation, indicating a peripheral lesion, or (3) no reaction to pure odors, but a response to all nonolfactory components, indicating central anosmia. Blocking of the trigeminal nerve and the taste nerves should permit better studies of olfactory qualities, especially since perception of taste qualities in the nose seems to be localized in the floor only.

According to Kindler, most traumatic anosmias are caused by changes within the cranial cavity, such as hemorrhage, or tearing of filaments, and sometimes by contrecoup. A malingerer will not be able always to give the same response to the same stimulant but will tend to suppress the reaction even when it is evidently wrong, as the nerves in question, namely, the trigeminal, show no other sign of involvement. A case of such malingering is discussed in detail. It is also advisable to have the patient localize by means of monorhinic examination. A specially

constructed cannula allows the examiner to bring the smell to one nostril while the other receives only air. Pure odors cannot be localized, but *Mischreizduftstoffe* give a definite localization. There will be numerous mistakes at the beginning of the examination, but the average of several readings will finally result in absolutely correct answers for normal persons, while that for malingerers will evidence gross mistakes.

LEDERER, Chicago.

Miscellaneous

PERIPHERAL NEUROPATHY AND TOXIC PSYCHOSIS WITH CONVULSIONS DUE TO SULFAMETHYLTHIAZOLE. C. GARVIN, Am. J. M. Sc. **200**:362 (Sept.) 1940.

Experience at the Cleveland City Hospital with sulfamethylthiazole (2-[paraaminobenzenesulfonamido]-4-methylthiazole) included toxic reactions similar to those with the other derivatives of sulfanilamide, namely, drug fever, dermatitis, granulocytopenia, hemolytic anemia and hematuria. In 1 case peripheral neuropathy appeared on the seventeenth day of treatment, and although administration of the drug was stopped, there developed a violent toxic psychosis with convulsions, delirium and fever. The patient became increasingly stuporous and died on the twenty-fourth day after institution of treatment. No neurologic abnormalities could be demonstrated, either clinically or at autopsy.

SHAMBAUGH JR. and JENNES, Chicago.

ACUTE NEPHRITIS: REVIEW OF SEVENTY-SEVEN CASES. J. HAYMAN JR. and J. MARTIN JR., Am. J. M. Sc. **200**:505 (Oct.) 1940.

Of 77 cases of acute glomerular nephritis, the disease followed infection of the upper respiratory tract, including the sinuses and the ears, in 75 per cent. The interval between the onset of infection and the renal complication was in the greater number of cases between one and three weeks. Streptococcus haemolyticus was the predominant organism in at least 44 per cent of the cases in which routine culture methods were employed, a figure which the authors remark as being much lower than the usual incidence reported in the literature.

SHAMBAUGH JR. and JENNES, Chicago.

ANTIPYRETIC ACTION OF SULFAPYRIDINE. P. B. BEESON and D. A. JANEWAY, Am. J. M. Sc. **200**:632 (Nov.) 1940.

The authors first present 3 clinical cases in which the drop in temperature following the administration of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) appeared to be due to an antipyretic action rather than to any effect on the infectious process. In each case the use of another drug of the sulfanilamide group failed to produce similar action. By experiments on rabbits the authors then demonstrate that sulfapyridine or one of its salts has a definite antipyretic action, far greater than the use of an equivalent amount of sulfanilamide or sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) or its salt. The authors offer the hypothesis that the mechanism of this action lies in increased dissipation of heat from the skin and describe interesting experiments as a basis for this concept. They suggest that the antipyretic action of sulfapyridine as compared with that of sulfanilamide or sulfathiazole "may be a point of some importance in the clinical evaluation of the effectiveness of these drugs."

SHAMBAUGH JR. and JENNES, Chicago.

SULFATHIAZOLE TREATMENT IN RESPIRATORY INFECTIONS. D. PEPPER and G. HAM, Am. J. M. Sc. **200**:784 (Dec.) 1940.

The authors report the cases of 52 patients with infection of the respiratory tract who were treated with sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole). Of these, 37 had typical pneumococcic pneumonia, with 2 fatalities, 11

atypical pneumonia, with 3 fatalities, and 4 bronchitis. All 5 fatalities were among the 11 patients over 50 years of age. Microscopic hematuria was present in 4 patients, and crystals of sulfathiazole or acetylsulfathiazole were found in the urine of 12 patients. In a patient on whom autopsy was performed concretions of acetylsulfathiazole were present within the collecting tubules of the kidneys. The authors stress that the greatest danger in the use of sulfathiazole or sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) lies in the recrystallization of the free or, more frequently, the acetylated drug, which is very insoluble, within the urinary passages. Sulfathiazole crystallizes out in the renal tubules, both in the experimental animal and in man, and sulfapyridine more frequently in the renal pelves and ureters. As the best means of avoiding the renal complications of treatment with either of these compounds the authors recommend an adequate fluid intake, at least 2,500 cc. daily, administration of an alkali, keeping of an accurate intake-output chart and daily urinalyses, especially during the first few days of treatment.

SHAMBAUGH JR. and JENNES, Chicago.

SULFATHIAZOLE IN BLOOD AND URINE. F. SUNDERMAN and D. PEPPER, Am. J. M. Sc. **200**:790 (Dec.) 1940.

The authors suggest that analysis for sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) be made on the blood serum rather than on whole blood, for whereas approximately 14 per cent of the drug is lost when added to whole blood, practically complete recovery is obtained when the drug is added to blood filtrates after protein precipitation. The solubility measurements of sulfathiazole and acetylsulfathiazole in urine and in dilute phosphate solutions show the acetylated form to be only about one-tenth as soluble as sulfathiazole and both to be far more soluble in alkaline than in acid solution. In urine with a p_H of 7.6 they were twice as soluble as in urine with a p_H of 5.6.

These data support the clinical observation that the occurrence of crystalline concretions in the urine appear to be lessened when the volume of urine is increased by adequate ingestion of water and its reaction is made alkaline. The authors conclude, therefore, that when crystalline concentrations threaten, as a result of sulfathiazole therapy, an effort should be made to keep the urine alkaline and to secure a large volume of urine.

SHAMBAUGH JR. and JENNES, Chicago.

HISTOPATHOGENESIS OF CEREBRAL ABSCESS. F. A. CARMICHAEL JR., J. W. KERNOHAN and A. W. ADSON, Arch. Neurol. & Psychiat. **42**:1001 (Dec.) 1939.

This article reviews the etiologic aspects of cerebral abscess and presents a detailed study of the evolution of the lesion, based on the gross and microscopic study of 60 brains. Of chronic infections of the head and face particularly predisposing to abscess of the brain, infections in and about the orbit are said most consistently to produce cerebral abscess. In the authors' series there were 3 cases of such infection.

IRVINE. [ARCH. OPHTH.]

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

JEROME STRAUSS, M.D., *President*

Regular Meeting, Jan. 5, 1942

Air Pressures in the Nose and Maxillary Sinus Under Normal Conditions and in Disease. DR. DELBERT K. JUDD, Chicago.

A preliminary report is presented on investigations of fundamental problems related to the etiology of sinus infection and the principles underlying methods of therapy. Some previous experimental studies were repeated and new investigations carried out. The investigations included: (1) comparisons of pressures in the nose and paranasal sinuses during physiologic processes, such as quiet respiration, blowing and sniffing, both under normal conditions and in disease; (2) observations on the effect on pressures within the sinuses of (*a*) mass suction, (*b*) irrigation and (*c*) combined suction and irrigation.

Under normal conditions pressure equilibrium is maintained at all times between the nasal and the sinus cavities. Blowing causes greatly increased pressure in both the nose and the sinuses. During the act of sniffing there are considerable but equal decreases in pressure in the nose and in the sinuses. When partial obstruction of the ostium is present from any cause a time lag occurs before the peak pressure in the sinus equals the pressure in the nasal fossa. The new pressure within the sinus tends to be maintained until an exaggerated opposing pressure is produced in the nose. The time lag in establishing pressure equilibrium is a direct indication of the amount of obstruction at the ostium and of impediment to the efficiency of the ciliary stream. With complete obstruction of the ostium there will be no alteration of the pressure existing in the sinus regardless of varying pressures which may be set up within the nose.

The relation of sinus and nasal pressure changes under different pathologic conditions is demonstrated by means of curves projected on the screen.

Observations on sinus pressures were made also under the following conditions:

1. Simple mass suction. The method is shown to be extremely inefficient in removing secretions from sinus cavities except when the sinus is partially filled with air and the fluid overlies the ostium. Under such conditions some fluid is removed, but only a fraction of the sinus content. (These observations do not include those on interrupted negative pressure, which is the basis of displacement therapy.)

2. Simple irrigation (irrigation without addition of suction). The irrigation is accompanied by positive pressure within the nose and (if the ostium is patent) in the sinuses. Secretions may be removed from the nasal cavity but not from the sinuses.

3. Irrigation combined with suction. This is the principle used by nasal siphons and suction irrigators. On analysis, this method is a combination of mass suction and an irrigating stream. The effect on the sinuses is precisely that of suction and is open to the same limitations.

DISCUSSION

DR. O. E. VAN ALYEA: Dr. Judd has brought up to date some of the physiologic concepts which should guide one at present in the treatment of sinus disease. Some years ago O'Malley of London demonstrated that there is a negative and a positive pressure change in sinus cavities, based on inspiration and expiration. He carried the theory to practical application. He expressed the opinion that on inspiration there occurs a definite removal of secretions from the neighborhood

of the ostiums of the sinuses, due to pressure changes. It seems to me that this pulling effect is similar to that produced by a passing streamlined train. I have always felt that secretions carried to the ostium of the sinus by the cilia and retained by a small ostium are often pulled through the opening by an inspiratory blast. Hilding emphasized the involvement of ciliary action based on these changes. He showed that in rabbits when the anterior naris is sewed up there is interference with ciliary action. He sewed up one anterior naris of a rabbit, left it for three weeks and found after removal of the sutures that the mucosa of both nasal cavities showed fragmentation of the cilia and some destruction of the mucous membranes.

In another experiment, McMurray instilled iodized oil into both antrums of a human subject and blocked off one naris. Roentgenogram taken at intervals showed that the oil on the occluded side was unmoved for seventeen hours, while on the open side the oil had passed out of the ostium and reached the floor of the nose in five hours. After removal of the occlusion, the oil on that side started to move at the same rate of speed and also reached the floor in five hours. Negative pressure caused by inspiration is shown by this experiment to be of vast importance in the evacuation of heavy material from the sinuses.

Dr. Judd's conclusions regarding mass suction are important. Most otolaryngologists feel that mass suction is of little value in promotion of drainage of the sinuses and that the procedure is often harmful. Anything that can be done to improve understanding of nasal physiology is to be commended. This work of Dr. Judd's is instructive and timely and, consequently, highly commendable.

DR. ALFRED LEWY: The mechanical contrivance known as an injector has been used by engineers to produce a flow from adjoining cavities through a pipe, simply by passing a jet of steam or air through a small pipe past an opening, corresponding to the ostium of the nasal sinus. That is what one does when one blows the nose.

One point with regard to mass suction seems to have been lost sight of by Proetz and others. Suction may not remove a secretion from the sinuses unless the discharge lies at the opening. There is the question of the therapeutic effect of hyperemia induced by suction. This increase in blood supply is a good idea under certain circumstances, though probably contraindicated in the acute stages.

DR. THOMAS C. GALLOWAY, Evanston, Ill.: This work seems to have been carefully done, and the conclusions should be valid. I did not quite follow Dr. Judd's logic and shall be interested in studying the written article. I still think suction may be valuable, but it must be used logically and gently and with visualization of what one is going to do. If one takes a model and carefully turns the ostium in the direction in which the secretion will overlie it, with air above that, one has a guide by which one can certainly empty even thick secretion if the ostium is in the proper position. Of course, suction does not work in the absence of air in a sinus. It is obvious that it should not be used to the extreme of producing hyperemia or rupturing vessels.

What happens to the blood vessels with suction? Positive and negative pressures should increase the circulation of the blood and aid in proper nutrition of the cilia and cells. Also, increase of negative pressure in the sinuses or absorption of air by blocking the ostium might lead to edema and possibly to cupping action on the capillaries, producing considerable increase in pressure and possibly hemorrhage.

DR. WALTER H. THEOBALD: Years ago it was the accepted opinion that the maxillary sinus most frequently involved was the one which was inaccessible. Some years ago I felt that theory could be disproved because so many antrums could be entered either by natural or by accessory openings, and in a series of 100 cases I determined that 52 per cent had accessory openings. The beautiful explanation by Dr. Judd shows that the sinus most frequently involved is the one easily accessible; therefore, the sinus with an accessory as well as a natural opening is most frequently affected. The cannula frequently slips into an accessory opening when one thinks one is in the natural opening.

DR. G. H. MUNDT: I have often noted the frequency with which the maxillary sinus is involved on the side of the concavity of the septum, and I think this theory of Dr. Judd's does much to explain that. Undoubtedly, the pressure is an important factor in producing more frequent involvement on the side of greatest concavity.

DR. DELBERT K. JUDD, Chicago: In the time allotted it would be confusing and ineffectual to attempt a detailed consideration of the apparatus or the mass of observations connected with the work done. The examples presented by the curves and charts were thought to be sufficient to illustrate the purpose and conclusions of the investigation.

Dr. Van Alyea compared the nasal air stream to a streamlined train in illustrating a process frequently experienced when a large mass of mucopus has been partly extruded through the ostium so that a portion of it extends into the path of the air stream. Forceful efforts of sniffing and blowing hurl a swift current of air against the mass, moving it backward and forward and finally pulling or dislodging the entire mass from the sinus. However, this mechanism comes into play only when the cilia have expelled a sufficient amount of the mass through the ostium to be caught in the air stream.

The injector principle spoken of by Dr. Lewy, as it exists at the ostium, is comparable to mass suction and can be effective only to that extent.

Perhaps, as Dr. Lewy says, the hyperemia produced by massive suction is an important therapeutic measure. I respect the great amount of clinical experience on which Dr. Lewy bases his statement, and I have no reason to disagree. I should only emphasize the fact that mass suction, being difficult to control, may exceed the negative pressure which can be tolerated by nasal mucous membrane. According to Proetz, a negative pressure in excess of one fifth of an atmosphere causes hemorrhage, which is not always evident, since it may remain submucosal. If infection of the hematoma occurs serious complications are possible.

In answer to Dr. Galloway, I agree that a small amount of secretion can be withdrawn from a sinus by mass suction provided the fluid overlies the ostium when the negative pressure is applied. The amount of discharge recovered, however, is only equal to the volume by which the air in the sinus is increased by the decreased pressure. This is an application of Boyle's law.

Since nasal mucous membrane will tolerate a decrease of only one fifth of an atmosphere of pressure, the amount of fluid which can be obtained is certainly limited. Air must be present in the sinus before this mechanism will work. No secretion can be withdrawn from a completely filled sinus by mass suction. Other factors which hinder this type of treatment are secretions of high viscosity and a partially or completely blocked ostium.

I should like to add to Dr. Theobald's discussion of the accessory ostiums of the sinuses, a fact not generally known. The studies of Proetz negate the supposition that an accessory ostium will permit a through and through flow of air. Pressures in the nose act on both openings equally and simultaneously, thus maintaining equilibrium between them.

Labyrinthine Dropsy and Ménière's Syndrome. DR. JOHN R. LINDSAY.

This paper was published in full in the June 1942 issue of the ARCHIVES, page 853.

DISCUSSION

DR. WILLIAM E. GROVE, Milwaukee: This paper constitutes a notable contribution to the question of the pathology of the Ménière complex. No satisfactory explanation or description of the pathologic background of this condition appeared after Ménière's original description in 1867, until Hallpike described the pathologic changes in the temporal bone in 2 patients who died after intracranial section of the auditory nerve. The pathologic process he described was similar to that shown in Dr. Lindsay's case. Hallpike advanced the hypothesis that the attacks of vertigo were initiated by what he called "bouts of labyrinthine asphyxia," brought

about by rapid rises in fluid pressure through relatively small increases in volume of endolymph. He contended that only after enormous dilatation of the endolymphatic cavities would the relatively small increases in volume of endolymph operate to produce the typical vertiginous attacks.

In the typical case of the Ménière symptom complex vertigo, deafness and tinnitus are associated. The vertigo comes on in typical attacks; the deafness is usually progressive, and at some time or other tinnitus is present. Furthermore, there are long periods of remission in some cases, so long, in fact, that the original vertiginous attacks may be all but forgotten. In the report of his case, Lindsay failed to mention any history of vestibular symptoms, but these may have been evanescent and forgotten. With herniation of the endolymphatic wall of the utricle into the ampullated ends of the semicircular canals noted in the pathologic specimens, it is inconceivable that this patient should never have had any vestibular symptoms. It may be true, however, that the increase in endolymphatic pressure and the effects on the sensory epithelium exerted thereby were so slow and gradual that vertiginous attacks were not set up in this case. It may also be true that the sensory epithelium of the vestibule and the semicircular canals may be more resistant to prolonged, but gradually increasing, pressure than that of the organ of Corti. Crowe also called attention to the fact that some of the cases of gradually increasing deafness may, in fact, be atypical cases of Ménière's disease.

Whether all cases of the Ménière complex would present the same pathologic picture which Hallpike and Lindsay have described is hard to say. As Lindsay points out, patients do not die of this condition and the temporal bones are not often obtained for pathologic examination.

That hydrops of the labyrinth might explain the symptoms of Ménière's disease is apparent from the previous observations of Dederding, Furstenberg, Crowe and Hallpike. Whether this hydrops is due to disturbance of water metabolism, as was assumed by Dederding, to disturbed sodium metabolism, as proposed by Furstenberg or to some other as yet unknown etiologic factor is not clear.

However, it is erroneous to say that dropsy of the labyrinth will explain the symptoms in all cases, for if such were the fact the symptoms would disappear after intracranial section of the auditory nerve or total destruction of the sensory epithelium, as practiced by Mollison, Berggren, Putnam and others. The symptoms of vertigo and tinnitus do not always completely disappear after these procedures. It is my contention, therefore, that whatever etiologic factor produces the hydrops probably also produces a "waterlogging" of the central cochlear, and particularly the vestibular, nuclei. There is no proof of this, except by deduction.

This work of Lindsay is extremely important in corroborating the observations of Hallpike and others, and it is to be hoped that in more cases of Ménière's disease the temporal bones may become available for pathologic study.

DR. GEORGE SHAMBAUGH JR.: This interesting and instructive paper is a real contribution to knowledge of Ménière's syndrome. Dr. Lindsay's observations are almost in complete accord with those I made in 1935 and 1936 on diplacucis and fluctuating deafness in this condition. He did not mention that the deafness is a low tone nerve deafness, but the audiograms in his cases show that hearing for the tones of low pitch are involved as much as or more than that for the tones of high pitch, contrary to the usual nerve deafness, in which the high tones are involved more than the low ones.

As I pointed out in 1936, I believe there is a syndrome of low tone fluctuating nerve deafness with diplacucis, in which the prognosis for hearing is relatively good if one can find an etiologic factor. Food allergy is a factor in many cases. I have a series of cases in which elimination of food allergy has resulted in considerable improvement in hearing as well as in vertigo. It seems to me that allergy may explain the hydrops of the labyrinth better than anything else. Allergy causes localized edema, with outpouring of fluid into the tissues, and the histologic changes in the cochlea, it seems to me, could be explained on this basis.

DR. JOHN R. LINDSAY: This patient showed low tone deafness with symmetric and normal upper tone limits, a normal reaction to the Rinne test and relatively

good bone conduction, which fluctuated with air conduction. Mygind and Dederding stated that this constituted a typical picture of Ménière's disease. In the 2 cases I presented previously there was greater loss for the high tones. The picture in the present case is probably a true one for Ménière's disease. The patient was under care for one year for attacks of vertigo before auditory disturbance developed.

Regarding the explanation of low tone deafness, the nerve apparatus, so far as could be told by histologic methods, appeared to be in good condition. Therefore, I am inclined to agree with Mygind and Dederding that this is due to a disturbance of conduction in the fluid system. They did not know there was hydrops of the labyrinth, but I think its presence bears out their conception of inner ear conduction deafness rather than nerve deafness. This theory better explains the fluctuations. When fluid is increased deafness is greater, and vice versa. It seems more reasonable to explain it on that basis than to assume that such wide fluctuations could occur week after week on the basis of nerve degeneration. I have tried inflating the tube repeatedly when patients felt that an attack was coming on, without results.

Of course allergy must be considered, but in view of the great number of patients with allergy and the infrequency of Ménière's disease, the percentage in which the two conditions appear to be related is extremely small.

On that basis my colleagues and I have been trying to produce vertigo in animals. We have been inducing allergic reactions in the middle ear, but so far have not been able to obtain nystagmus. The work is not yet finished. This case was included as an instance in which the pseudo-Ménière syndrome persisted for a year and was followed by the complete syndrome.

I want to thank Dr. Grove for his discussion. As to the cause of the functional disturbance, Hallpike based his explanation on increased pressure. That explanation was never satisfactory; moreover, as Crowe pointed out, the labyrinthine fluids are subjected to enormous pressures in certain diseases of the central nervous system without deafness or vertigo. It is impossible to get pressure in the endolymph much greater than that in the perilymph because of the extreme thinness and nonelasticity of the membranes as long as any perilymph remains in the labyrinth.

Acoustic Movements of the Human Sound Conduction Apparatus. Dr. H. F. G. Kobrak, Gary, Ind.

Direct observation of the conducting mechanism during acoustic stimulation is difficult in man for a number of reasons. The anatomic structures are minute and not readily accessible. The amplitudes of the movements are small and, finally, the vibrations so fast that the human eye cannot record them. A number of technical improvements have been made to overcome these obstacles.

By using an appropriate optical system all important anatomic structures can be sufficiently enlarged. Mirrors on the vibrating parts provide a suitable means for objective observations. The value of the moving picture camera as a recording apparatus is demonstrated. The picture begins with the experimental setup. It shows a machine which produces slow changes in air pressure of sinus form. These changes in pressure can be considered as tones below the lower threshold for hearing. These slow movements of the ear drum, the ossicular chain, the stapes and the round window membrane are shown as they appear in fresh cadaver specimens. Acoustic vibrations are next seen. With both ordinary, 16 frame exposure and slow motion photography acoustic movements of the ossicles are demonstrated on cadaver specimens.

The next development of the method is the use of stroboscopic light (120 interruptions per second). Various tones near 120 vibrations are directed into the outer ear. The clear visibility of the amplitude of the ossicles is striking.

The next part of the film deals with the acoustic stapedius reflex in cases of perforation of the ear drum and in a case in which radical mastoidectomy had

been done. The tendon of the stapedius muscle and the head of the stapes are visualized.

The last part of the moving picture shows the ear drum of a normal person. A tone is directed into the outer ear. No movements of the membrana tympani can be seen. If, however, stroboscopic illumination is substituted for ordinary light, definite movements of the membrana tympani are demonstrable. With this method it has been possible for the first time to see directly the acoustic oscillations of the ear drum.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION OF OTOLARYNGOLOGY

KARL M. HOUSER, M.D., *Chairman*

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Regular Meeting, Jan. 21, 1942

Value of Roentgenology in the Diagnosis and Treatment of Some Otolaryngologic Conditions. DR. W. E. CHAMBERLAIN (by invitation).

The interpretation of roentgenograms of the sinuses is often difficult, but, in my experience, it is not difficult to speak with assurance when the roentgenogram appears normal. In other words, I have a sense of adequacy and security when I can state: "The roentgenographic appearance is normal." It is frequently said that acute sinusitis escapes the powers of observation of the roentgenologist and that an acute inflammatory condition may exist when the roentgenogram is normal. I do not agree with this. I have made roentgenographic examinations of healthy adults and have brought the films out of the storage vault for purposes of comparison when, at a later date, acute coryza had developed. Invariably in my experience the acute inflammation of the sinuses has been visualized.

Carcinomatous infiltration frequently manifests itself in recognizable changes in the roentgenogram, but I have had the experience of making a diagnosis of carcinoma of a maxillary antrum or of the ethmoid region, only to have the case turn out to be one of inflammation. In other words, inflammatory lesions of unusual severity may occasionally simulate carcinoma. Likewise, when the destruction of bone accompanying carcinomatous inflammation has not yet reached a point at which the roentgenographic appearance is characteristic or recognizable, a carcinoma may be mistakenly diagnosed as an inflammatory lesion. In such instances, however, one usually suspects the possibility of carcinoma from the localization of the changes (severe changes in one locality without the generalized distribution to be expected), even when a definite diagnosis of neoplasm cannot be made.

I should like to emphasize the importance of making the roentgenograms with horizontal roentgen beams as often as possible. Whenever an examination is made with the roentgen beam vertically disposed, the opportunity to visualize a fluid or pus level, if present, is thrown away. The easiest way to utilize the horizontal beam is with the patient erect, but even when he is bedridden or confined to the stretcher, the beam can be utilized by propping up the film on its edge and projecting the rays horizontally through the patient's head.

(Illustrations showed planigraphic sections in two coronal planes 4.5 and 5 cm. from the table top, with the patient prone.) These two planigrams were made only 5 mm. apart. They show how very "thin" the sections can be made with this new method. The section 4.5 cm. from the table top barely enters the maxillary antrums, while the section at 5 cm. reveals the outlines of the antral

walls nearly completely. The demonstration of the bony structure within the turbinates is extremely clear. This is not visualized in ordinary roentgenograms.

(Illustrations of four planigraphic sections through the sinuses of a patient with a submucous cyst in the right maxillary antrum, taken 6, 7, 9 and 11 cm., respectively, above the table top, with the patient prone.) As yet the laboratory with which I am associated is not equipped with apparatus to make planigraphic sections with a horizontal roentgen beam. For this reason, complete information with respect to fluid and pus levels cannot be obtained. This is not extremely important, because planigraphic studies are practically always supplementary to a routine nonplanigraphic examination. In the present case the 6 cm. section shows the submucous cyst in the right maxillary antrum fully as clearly as it could have been shown with iodized poppyseed oil, instead of air, as the contrast medium. I believe that iodized poppyseed oil will probably be used much less frequently in examinations of the sinuses now that planigraphy is available. The 7 cm. section coincides with the ostiums of the maxillary antrums. The section is so 'thin" that these ostiums would not have been shown had I focused on a plane even a few millimeters closer to or farther from the table top. The detail in the walls of the ethmoid cells in the 7 cm. section is noteworthy. The 9 and 11 cm. sections show, respectively, the ventral and the dorsal portion of the sphenoid sinus. Note that in both these sections the ethmoid cells are completely blurred out because they are ventral to the planes in which the sections were made.

(Illustrations of standard and unusual angles of projection for roentgen study of the mastoids.) These roentgenograms show the advantage of many different angles of projection, including the "occipital projection" (central ray of the roentgen beam directed from the hair line at the top of the forehead to the dorsal lip of the foramen magnum, with the film at the nape of the neck), "high angle" vertex projection (central ray of the roentgen beam projected from the region of the hyoid bone to the vertex of the cranium), special oblique projection of the petrous portion of the temporal bone in either the Stenvers or the Blackstone position and high angle vertex projection with the planigraph.

(Planigrams and conventional roentgenograms of the larynx and the upper portion of the trachea.) Planigraphy impressed itself on the medical profession in connection with the anteroposterior examination of the larynx long before its usefulness was proved in any other field. Ability to blur out the details in the cervical portion of the spine, with clearer visualization of laryngeal structures, in a thin plane selected by the roentgenologist provided for an entirely new method. So far as the chief diagnostic contribution of the roentgenologist is the visualization of gross anatomy, both normal and morbid, planigraphy represents a tremendous advance. (Planigrams showing laryngeal carcinoma, unilateral paralysis of the recurrent laryngeal nerve, the normal larynx, subglottic extensions of tumors, an unusual neurofibroma in the subglottic region, foreign bodies in the cervical region, tumors of the thyroid, etc., were demonstrated.)

The Otolaryngologist and Roentgenology. Dr. Karl M. Houser.

The great value of roentgenology in determining treatment suitable for various otolaryngologic conditions is stressed, with special emphasis on the advantages to be gained from consultation of the roentgenologist and the otolaryngologist after the roentgenologic examination is completed. With regard to indications for surgical intervention, the roentgenologic findings may be somewhat misleading, and the decision to operate should rather be based on clinical observations. Necrosis is, however, usually an indication for surgical intervention. It must be kept in mind that the roentgenologic changes may be due to permanent changes in the tissues from preexisting disease or to allergy. The roentgenologist preferably should submit his report and leave interpretation to the otolaryngologist after correlation with his own observations. In cases of sinus conditions, the roentgenologic report is likely to be more favorable than the rhinologic report. The great discrepancy in reported results of treatment may be due in part to the fact that acute conditions recover spontaneously whereas chronic conditions are

extremely resistant to treatment and, accordingly, the results obtained will depend entirely on the type of condition treated.

Roentgen therapy may be of value in selected cases of sinus disease. In my experience it has been disappointing, and I feel it should be tried only in cases in which conservative treatment fails. Among the conditions for which roentgen therapy has been employed, good results have been reported in cellulitis, erysipelas, furunculosis and eczema. I have not found roentgen therapy of value for shrinking of nasopharyngeal lymphoid elements or for the chronic deafness of tinnitus unless it is due to tubular obstruction or congestion. The method is no longer recommended for treatment of infection of the faucial tonsils.

DISCUSSION ON PAPERS BY DRS. CHAMBERLAIN AND HOUSER

Dr. George M. Coates: I wish to compliment the speakers on this excellent presentation. Frequently patients from another city present themselves for examination, bringing their roentgenograms with them. In many cases the report does not accompany the film. In the majority of cases I am not satisfied with my interpretation of the roentgenograms, and therefore I seek consultation with my roentgenologic friends. They, also, often frankly admit that they have the same difficulty. Therefore, a new roentgenographic study is necessary. The planigraphic technic is proving to be a valuable aid to the otolaryngologist, but it is not well to be guided entirely by the roentgenographic findings in any case. I am reminded of an elderly physician who told me that in a suspected case of acute mastoiditis he always followed the advice of the roentgenologist; if the advice was to operate he always operated, as the roentgenologist was always right. The patient's symptoms were unimportant, he said!

Dr. Harry P. Schenck: Dr. Pendergrass mentioned 2 cases of mucocele of the maxillary sinus in both of which bizarre symptoms were presented. In each instance the presence of mucocele was discovered by roentgenograms, for the normal antrum failed to transmit light as well as did the antrum containing the mucocele. Both patients were physicians. One had had attacks of vertigo for ten years prior to removal of the mucocele. The attacks were severe but did not result in falling. An increasing loss of acoustic acuity on the side of the lesion was noted with the audiometer. The second patient had sweating over the side of the head of the involved antrum and over the opposite side of the thorax. He had frequent unexplainable attacks of weakness and a sense of fear and terror in crowded places. Both patients were entirely relieved of symptoms after the mucocele was removed by the Caldwell-Luc approach, but the deafness in the case of the first patient did not improve, although it has not progressed further in the past five years.

The bizarre symptoms arising from mucocele in the maxillary sinus are difficult to explain, but Larsell, Barnes and Fenton showed that constriction of the vessels could be obtained after stimulation of the nasal mucosa on the side on which the maxillary division of the fifth cranial nerve had been sectioned. They expressed the belief that this indicated that other afferent fibers are involved in the innervation of the mucosa of the nose besides those carried by the maxillary division of the fifth cranial nerve and that these fibers cause vasoconstriction; such fibers are carried in the posterior nasal, the great superficial petrosal and possibly the deep petrosal nerve. The vasomotor response was abolished by section of the spinal cord at the level of the sixth cervical segment or by section of the cervical part of the sympathetic trunk between the middle and the inferior cervical ganglion.

Roentgenograms are especially valuable to the clinician when the clinical picture is apparently normal. Thus, a small antrum with thick bony walls may be entirely normal, while a large antrum on the opposite side may be filled with diseased mucous membrane. Both may be transilluminated equally well. The roentgenologist can also differentiate between abnormalities of the sinuses due to allergy and those due to chronic infection. In allergy there occurs absorption of bone, so that the partitions of the ethmoid cells are decalcified. The malar bone and the zygomatic arch are seen too clearly through the antrum. Increase in blood

supply to the part, together with pressure from the polypoid tissue, is followed by decalcification of bone. In chronic infection osteitis and sclerosis involve the walls of the sinuses. When the antrum and the ethmoid cells are affected the osteitis extends to the greater wing of the sphenoid bone, the superior maxilla and the malar bone.

I do not believe that the roentgenologist can detect the early changes in acute infection, but he can detect far better than the rhinologist the changes due to chronic infection. It is impossible as a rule to differentiate between pus, the mucous membrane and the normal secretion of the mucous membrane.

DR. BENJAMIN H. SHUSTER: It is hardly fair to expect an otolaryngologist to discuss a talk by a roentgenologist on roentgen diagnosis. One, however, cannot help admiring and commenting on the beautiful demonstration given tonight. It takes a great deal of experience and imagination to be able to place a head and photograph it so as to give detailed information at certain desired parts. This leaves no doubt that a roentgenologist should be not only an excellent technician but a student. It is to be regretted that the essayists did not consider the question of treatment. An otolaryngologist could then be in a position to discuss the subject. I have had some experience with treatment, but it has not been altogether favorable.

I recall a patient, a boy about 15 years of age, with acute mastoiditis and what was suspected to be petrositis. He had been sick for about a week, with high fever, profuse discharge from the ear and pain in the eye. This occurred at a time when roentgenographic technicians were not yet acquainted with the proper angle of taking base roentgenograms. The technician therefore failed to obtain the proper angle and repeated the exposure several times. By the time the boy had been roentgenographed from many angles the temperature dropped to normal, the discharge from the ear diminished to almost nothing and the pain in the ear disappeared. I believe that the terrific exposure to roentgen rays relieved the mastoiditis and cured the patient. I have received similar impressions in other cases of acute mastoiditis, but not quite such a decided one. I cannot say the same thing with respect to chronic infection of the mastoid, otitis media or sinusitis. There are conditions in which one could expect a good result from roentgen treatment. Of course, treatment of malignant growths has given favorable results in a considerable number of cases.

DR. ROBERT J. HUNTER: The roentgenograms taken by Dr. Pendergrass with the patient in the upright position are very good, and I wish to compliment him on these especially, although all of his illustrations are splendid. The planigrams taken by Dr. Chamberlain are convincing. With such plates one can determine before operation the cases of mastoiditis in which the external layer of cells derived from the squamosa is clear and the internal layer derived from the petrosa is involved. One so often hears that the mastoid showed nothing until the operator's knife was deep, where the disease was severe.

Mortimer described how he arrived at the conclusion that atrophic rhinitis is associated with hormonal deficiency by searching for human skulls with characteristics similar to those of the skulls of rats which had been operated on to make them deficient in estrogen. I had hoped to examine the roentgenograms of patients with atrophic rhinitis or those with the typical facial appearance of underdevelopment of the superior maxilla and malar bone to see whether any anthropologic measurements of interest could be obtained. I had thought also of making these measurements directly on the patient and then trying to coordinate them with the roentgenograms and the physical appearance of the nose. If roentgenologists were acquainted with these measurements they could report the cases in which the facial bones had failed to develop in proportion to the calvarium. There is such a variation between the clinical and the roentgenologic diagnosis in many cases of sinusitis that it is probable that some of the abnormalities in the roentgenogram are the result of former infection or of some constitutional change rather than the immediate result of sinusitis. I refer especially to the cases in which the roentgenologist reports that the ethmoid cells are involved.

DR. MATTHEW S. ERSNER: The employment of roentgenologic studies for diagnosis is indispensable. From a therapeutic standpoint, roentgenography should be considered as an aid rather than a routine procedure in the treatment of benign otorhinologic conditions. Irradiation is not a panacea for such conditions and is not effective in all cases. It is frequently instituted haphazardly or empirically in cases not suitable for such treatment, and in such instances, naturally, the end result is failure. However, if roentgen therapy is employed rationally and in selected cases, for example, in those of cellulitis of the external auditory canal, eczema of the external auditory canal and various types of sinusitis, as an adjunct to the routine treatment, the desired effect may be obtained. It is essential, therefore, that there be complete cooperation between the roentgenologist and the otorhinologist, so that results and controls may be evaluated.

Planigraphy has a great deal to offer, and with improvement in the technic and further knowledge with regard to interpretation, seriatim studies enable one to visualize minutiae and details of structures in an involved area.

Several precautions should be taken when a patient is referred for roentgen study. In cases of the hyperplastic and the allergic type of nasal mucous membrane, one should administer epinephrine and atropine one-half hour before roentgen studies are made, so that these drugs may act on the vasomotor system and help create dryness and uniform decongestion of the nasal mucous membrane. Otherwise, the edematous tissue will cast a shadow which may be misinterpreted.

In 1934 I called attention to the fact that roentgen studies should not be made immediately after decongestion of the nasal mucous membrane, as under these circumstances the sinuses have evacuated themselves and the mucous membrane will not show any thickening. On the other hand, if roentgenograms are taken several hours after the shrinking of the mucous membrane, there may be vasodilatation with secondary edema of the mucous membrane, shadows thus being cast which do not exist. It is also true that after infection one must be careful in the interpretation of thickened tissue undergoing resolution, which from a roentgenologic standpoint might be considered as pathologic tissue.

DR. W. E. CHAMBERLAIN: The intrapharyngeal roentgenograph for study of the larynx is of no further interest to the roentgenologist now that he has the planigram.

The work of Dr. S. J. Crow, in Baltimore, on application of radium therapy in the nasopharynx for prevention of deafness deserves one's support and continued interest. Dr. Crow insists that he has no answer to his problem and that he must wait ten or more years for a final conclusion. His well considered experiment consists in irradiating the nasopharynx of the child who has hypertrophic lymphoid elements that close, or partly close, the eustachian tubes and then waiting ten years or more to determine whether the incidence of deafness is decreased through this modality. It appears that the experiment is well planned and the method altogether laudable.

I am extremely skeptical as to the value of roentgen therapy for disease of the sinuses. No real proof has yet been produced by any one. In my opinion roentgen therapy of the sinuses is resorted to about ten times as frequently as is justifiable, in view of the fact that many of the patients treated would have made complete recoveries without such therapy. The doses resorted to are usually sufficiently high to produce tissue changes of a permanent and irreversible type. These are to be avoided. The dose should be extremely low, and the treatment should be limited to cases in which one is reasonably certain that spontaneous recovery or recovery from less potentially harmful methods of treatment may not be expected.

Planigraphy will be of great value in the study of osteomyelitis of the frontal bone. My opinion in this matter is based not on actual experience with this condition but on observations on similar conditions in the third lumbar vertebra and other parts of the spine. In some cases of suppurative vertebral spondylitis planigraphic studies revealed important details that were not even suggested by conventional roentgenograms.

Book Reviews

Elimination Diets and the Patient's Allergies: A Handbook of Allergy. By Albert H. Rowe, M.D., Lecturer in Medicine, University of California Medical School, San Francisco; Consultant in Allergic Diseases, Alameda County Hospital, Oakland, Calif. Price, cloth, $3. Pp. 264, octavo. Philadelphia: Lea & Febiger, 1941.

The author discusses the many phases of clinical allergy in a clearcut and comprehensive manner. The textbook is chock-full of practical information. Nine pages are devoted to the subject of inhalant allergens.

The fallibility of skin tests, test negative diets, modified elimination diets and food addition diets is fully considered. Important points in the diagnosis of allergy are brought out briefly but pointedly, so that they may be readily adapted to any given clinical case at hand.

The causes of allergy are fully considered in seventy-one pages, and the discussion is a thorough one.

Under the heading of clinical allergy and its control, the author discusses in fair detail allergy in the various systems.

The discussion of the management of allergy by elimination diets is thorough and most useful. Following the final chapter is an appendix, wherein the author deals with such subjects as vitamins, daily nutritional requirements and maintenance of weight. Instructions for the establishment of environmental control, a questionnaire and a history form for allergic patients, general directions for pollen therapy, a food diary and other features are included in this appendix.

The author has a rich background of experience, reflection and deliberation in this particular field and presents to the members of the medical profession the important points of this background in the form of a clear, concise and lucid textbook.

He has done more than many authors to bring the important practical points quickly and forcibly to the fore by the use of italics, so that any physician can quickly grasp the message and use such thoughts as may be applicable to any given case. Indeed, it may well be said that this textbook can be used to advantage "by him who runs."

Otolaryngologists encounter varying states of allergy in their daily practice, and whether one refers allergic patients or treats some phase of allergy himself, he is certain to obtain much authoritative help by consulting this book.

This textbook is strongly recommended to all otolaryngologists, indeed to all specialists in the medical and surgical fields, and the author deserves the gratitude of all for his help in the solution of this and that baffling problem which stand in the way of the ultimate hoped-for successful conclusion.

Nursing in Diseases of the Eye, Ear, Nose and Throat. By the Staff of the Manhattan Eye, Ear and Throat Hospital. Price, $2.50. Pp. 313, with 94 illustrations. Philadelphia: W. B. Saunders Company, 1942.

This little book of 313 pages has been written by nationally known chiefs of service of the Manhattan Eye, Ear and Throat Hospital of New York city; that it is in its seventh edition since 1910 speaks in no uncertain terms for its popularity. The authors are Dr. David H. Webster, surgeon director of the ophthalmic department, and Dr. John R. Page, Dr. Francis W. White, Dr. Robert E. Buckley and Dr. Daniel S. Cunning, surgeon directors of the department of ear and throat diseases. In addition, chapters are contributed by Dr. Alfred G. Langmann, consulting pediatrician; Dr. Andrew A. Eggston, pathologist, and Hazel Emmett, R.N., superintendent of nurses.

In this, the seventh edition, six new chapters have been added to the already well covered subjects, bringing them up to date in regard to advances in diagnosis, new discoveries and changes in treatment of diseases of the eye, ear, nose and throat, written especially for the instruction and guidance of members of the nursing profession. Old chapters have been adequately revised.

New chapters are included on the treatment of syphilis of the eye, ear, nose and throat; the use of tuberculin in diagnosis and treatment in ophthalmology; the drugs of the sulfanilamide group; allergy; vitamins, and clinical pathology in ophthalmology and otolaryngology. The chapters on external diseases of the eye; tracheotomy, and bronchoesophagology have been rewritten for this edition, while new sections on foreign protein therapy, postoperative duties, orthoptic training and acute laryngotracheobronchitis are added.

While this book is designed and written primarily for the nursing profession, for which it is admirably adapted, the wealth of material presented will cause it to appeal almost equally to medical students (even graduate students in the specialties covered), to interns and residents and, last but not least, to the general practitioner. The simplicity of its language and style, the concise directions and appropriate illustrations make for easy reading and ready comprehension. It outlines chiefly the methods employed in the Manhattan institution, but these differ only in minor details from those in common use elsewhere, while the distinction of the authors gives them authority.

This reviewer has read this new edition of a standard nursing work with pleasure, admiration and profit and recommends it unreservedly to the professional classes mentioned in the preceding paragraph.

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American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 11-14, 1942.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James G. Dwyer, 375 Park Ave., New York.
President-Elect: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Edward J. Whalen, 750 Main St., Hartford, Conn.
Southern.—Chairman: Dr. Francis B. Blackmar, Swift Kyle Bldg., Columbus, Ga.
Middle.—Chairman: Dr. Voss Harrell, Hofman Bldg., Detroit, Mich.
Western.—Chairman: Dr. Irving M. Lupton, Medical Arts Bldg., Portland, Ore.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.